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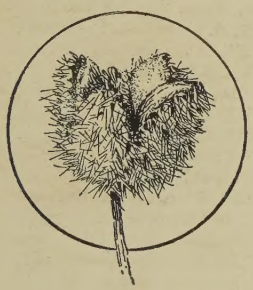
Department of Plant Pathology

ENDOTHIA CANKER OF CHESTNUT



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By P. J. ANDERSON AND W. H. RANKIN

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CANKER ON TRUNK OF CASTANEA DENTATA CAUSED BY THE FUNGUS ENDOTHIA PARASITICA. YELLOW TENDRILS OF PYCNOSPORES SHOWN AROUND MARGIN

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CONTENTS

PAGE

Hosts 533
Economic value of chestnut 535
Soil requirements 536
Natural reproduction 536
The disease 537
Name 537
History 538
Origin of the epiphytotic 539
Spread in the United States 539
Distribution in New York State 542
Range of the chestnut in New York State 543
Conditions in 1911 543
Conditions in 1913 545
Economic importance 545
Symptoms 545
Etiology 550
Name of the causal organism 550
History of the pathogen 554
Morphology 557
Stromata 557
Pycnidia 559
Pycnospores 560
Perithecia 561
Asci 562
Ascospores 562
Mycelium 563
Pathogenicity 563
On *Castanea dentata* 563
On other species of Castanea 563
On trees outside the genus Castanea 564
Life history 565
Germination of pycnospores 565
Germination of ascospores 567
Vitality and longevity of the spores 567
Pycnospores 567
Ascospores 569
Inoculation and infection 569
The inoculum 569
Necessity of a wound 569
Agents that produce the wounds 570
Age of tree and parts of host affected 572
Effect of season 573
Development of the canker 573
Rate of growth of mycelium 574
Vitality of mycelium 575
Development of pycnidium 576
Time of production of pycnospores 577
Development of perithecium 578
Time of development of perithecia 580
Ejection of ascospores 580
Saprophytic growth 581
Dissemination 582
Man 583
Insects 585
Rain 586
Birds 587
Wind 587
Other animate agents 589

PAGE

Pathological histology 589
Cultural studies 590
Media 590
Isolation 590
General cultural characters 591
Sterilized twigs 591
Potato agar slants 592
Relation of light 592
Relation of temperature 592
Tannic acid 593
Ecologic relations 593
Control 595
Methods that have been advocated 595
General discussion of control measures 605
Exclusion measures 605
Eradication measures 606
Protection measures 608
Immunization measures 608
Recommendations for timber owners in New York State 609
The outlook 610
Acknowledgments 610
Bibliography 611

ENDOTHIA CANKER OF CHESTNUT

P. J. Anderson and W. H. Rankin [1]

(*Received for publication March 27, 1914*)

HOSTS

The Endothia canker was first discovered by Merkel (1906), on the American chestnut (*Castanea dentata*), in the New York Zoological Park during the summer of 1904. Metcalf (1908 a) states: "Observations made by the writer during the past year indicate that all varieties and species of the genus Castanea are subject to the disease except the Japanese varieties (*Castanea crenata* Sieb. and Zucc.)." In the same year Metcalf (1908 b) states that the Japanese varieties are in general resistant, and Murrill (1908 a:27) reports having found the canker on a Japanese chestnut and on the chinquapin (*Castanea pumila*) in the New York Botanical Gardens. Pantanelli (1911), Metcalf (1912 b:76), and Morris (Clinton 1911:725) give evidence that varieties of the European chestnut (*Castanea sativa*) are also subject to this disease. Morris (Clinton 1913:376) gives his observations on orchard varieties as follows: Corean, Chinese, and northern Japanese varieties were not attacked, although they had grown for five years alongside diseased trees, while southern Japanese varieties were attacked; but the Corean, Chinese, and northern Japanese varieties were attacked after having been grafted on American stocks. A small number of the eastern and the western chinquapins were unaffected except for one branch of a single tree. Many points that are essential for determining the real value of these observations as establishing the immunity or the susceptibility of these varieties are unfortunately not given. Fairchild (1913) reports having found the disease in northern China on a species of Castanea, probably *C. mollissima*.

Van Fleet (1914) records many observations concerning the spread of the canker in the breeding-plots at Washington, D. C., where he has been breeding chestnuts experimentally since 1894. Nearly all the trees having *C. americana* in any combination have disappeared. Recently Van Fleet has worked with selections made from self- and chance-pollinated individuals of the chinquapin and Asiatic types. He has obtained many precocious hybrids from these crosses. He reports his observations on the resistance to the canker shown by these hybrids as follows:

"Seedlings of Paragon chestnut, the best variety of the European type, pollinated with our native species, attained an average height of

[1] Names of authors arranged alphabetically.

twenty-five feet and were bearing excellent nuts when attacked in 1910, but all have succumbed. The crosses of Asiatic and native, fewer in number, showed greater resistance but all have been seriously affected. The chinquapin-European hybrids are readily affected but have great recuperative powers, bearing nuts the second year on suckers springing from the bases of diseased stems. Chinquapin-native crosses with the exception of the Rush chinquapin — a probable natural hybrid found wild in Pennsylvania — appear very susceptible and do not as readily recover. The wild chinquapin itself appears measurably resistant, several individuals, including two Rush chinquapins, thriving for years with no signs of disease though constantly surrounded by infection.

"The Asiatic chestnuts, and the chinquapin-Asiatic hybrids, are plainly highly resistant. Few have shown any appearance of infection and when noticeable the injury is quite local in character. Second generation seedlings of chinquapin-crenata crosses show no disease at all though always exposed to infection."

Morris (1914) reports that five trees of the species *C. mollissima* have not been affected, although American trees have died all around them. Specimens of *C. alnifolia* also have remained free from the disease.

In brief, it may be said that there is no species of Castanea which is wholly immune. Some varieties show marked resistance, especially Asiatic varieties, and Metcalf (1914) claims apparent immunity for certain strains. On the other hand, no species outside the genus Castanea is known to be seriously affected.

The species of Castanea are confined to the northern hemisphere and are widely distributed through their range. Schneider [2] states that there are five species which are very closely related one to the other, in part probably representing only geographical varieties. The following species are recognized: *C. crenata*, of Japan and Central China; *C. sativa*, of southern Europe and northern Africa, and eastward to Persia; *C. dentata* and *C. pumila*, of eastern and southern United States. A species *C. mollissima*, of which little is known, has been described from China.

Many varieties from Europe, Japan, and China have been introduced into the United States for orchard culture. Taylor [3] states that the European chestnut was introduced into this country in 1803 and the Japanese chestnut in 1876. Corsa,[4] speaking of the European and the Japanese species, states: "Both species can be grown in portions of this country, either as seedlings or as grafted trees on American stock. . . The majority of imported trees and seedlings raised in this country from

[2] Schneider, C. K. Handbuch der laubholzkunde 1: 156. 1906.
[3] Taylor, W. A. Bailey's Encyclopedia of American horticulture 1: 294.
[4] Corsa, W. P. Nut culture in the United States. U. S. Agr. Dept., Pomol. Div. 1896. (Unnumbered bulletin.)

imported nuts are injured or killed entirely by our severe winters. It is doubtful whether five per cent of the imported European chestnuts live long enough to come into bearing, but stocks raised from seed of the few exceptional hardy trees which do flourish here are generally hardy, and in this way a strain of European chestnuts has been secured that is well adapted to the climate of the Eastern States. . . . Trees from nuts imported from France and Spain have been fruiting for at least a half century near Philadelphia, Pennsylvania, and Wilmington, Delaware." These cultivated varieties are now being grown throughout the eastern and southern States, although the greater part of the chestnut-growing industry is confined to the States of Delaware, Maryland, Pennsylvania, New Jersey, New York, and Connecticut.

There are two native American species of Castanea, *C. dentata* and *C. pumila*. Sargent [5] gives the distribution of the forest species (*C. dentata*) as from " southern Maine to the valley of the Winooski River, Vermont, and southern Ontario, along the southern shores of Lake Ontario to southern Michigan, southward to Delaware and southeastern Indiana, and along the Alleghany Mountains to central Alabama and Mississippi, and to central Kentucky and Tennessee . . . . attaining its greatest size in western North Carolina and eastern Tennessee " (Fig. 77, page 542). *C. pumila*, as described by Sargent,[5] is found from southern Pennsylvania to northern Florida and west to Arkansas and Texas, is usually shrubby in the region east of the Allegheny Mountains and arborescent west of the Mississippi River, and is most abundant and reaches its largest size in southern Arkansas and eastern Texas.

ECONOMIC VALUE OF CHESTNUT

From an economic standpoint the American chestnut is by far the most important species. It is one of the main forest trees of New York, Connecticut, Pennsylvania, and the Allegheny Mountain region southward to Alabama. The cut of chestnut comprised about seven per cent of the total amount of hardwood timber cut in the United States in 1910. In 1907 the United States Bureau of the Census [6] reports: " Chestnut is a wood whose use for lumber has increased remarkably within the last few years. The total cut in 1907 was over three times as large and its total value over four and one half times as great as in 1900 . . . having a value of $11,130,547 . . . . nearly one half of the total cut was reported by Pennsylvania, West Virginia, and Tennessee. . . . In addition to being largely used for lumber, much chestnut is also cut into posts, poles, rails, and cross-ties, and used in the manufacture of

[5] Sargent, C. S. Manual of the trees of North America, pp. 220–222. 1905.
[6] The lumber cut of the United States, 1907. U. S. Census Bureau. Forest Products, No. 2 : 1–53. 1908.

tanning extract." For 1907 the value of the chestnut used in the latter products amounted to $1,619,785 for poles, about $3,500,000 for ties, $2,560,007 for tanning extract, and $377,880 for slack-cooperage. The total value for all chestnut used in the products reported amounted to $19,188,219 in 1907. The later reports show but little variation from these figures for the yearly consumption. Unfortunately value computations are omitted in the reports for 1910 and 1911. For 1909 the total value of the chestnut cut, according to the reports, amounted to $19,098,581.

Besides its commercial value the chestnut has been utilized in its natural range as a much-favored ornamental. Many large estates in New York, Pennsylvania, New Jersey, and Connecticut valued their chestnut trees very highly. As an orchard tree the varieties of the American and the foreign species are of relatively less economic importance. The chinquapin, except as a variety for nut production, is of little commercial value.

The chestnut tree is our most important source of tanning extract. We know of no rapidly growing species that could be economically substituted in case of its extinction. The result of this would inevitably be a material increase in the cost of tanning leather.

SOIL REQUIREMENTS

Concerning the soil requirements of the chestnut, Zon [7] states:

" Chestnut is not very exacting in its demands upon the nutritive substances of the soil, but requires that it be deep, fresh, loose, and moderately fertile. The development of chestnut seems to depend more on the situation and the physical conditions of the soil than on its chemical composition. A moderate amount of clay, though not enough to interfere with the looseness of the soil, together with some potassium and lime, suits the species best. . . .

" Chestnut is a deep-rooted species, which derives its nutrition from the lower layers of earth — a fact explaining its vigorous growth on exposures with poor surface soil."

NATURAL REPRODUCTION

Quoting further from Zon: " The conditions for the reproduction of chestnut from seed are very unfavorable. The presence of man, who has made a business of gathering and selling chestnuts, of hogs, which seek them greedily, and of coppice chestnut and other hardwoods, under whose dense shade the chestnut seedlings must come up, renders reproduction from seed almost impossible."

[7] Zon, Raphael. Chestnut in southern Maryland. U. S. Forestry Bur. Bul. 53 : 1–31. 1904.

The chestnut is noted for its ability to sprout from the collar after the tree is cut, forming a dense growth of coppice. Practically all the second and successive growths of chestnut in the Eastern States have come about in this way. This coppicing method of reproduction is followed in forestry practice to great advantage. The influence of different factors on the thriftiness of coppice growth are discussed by Zon.[8] Many persons have contended that this practice has devitalized the chestnut, making it more susceptible to diseases; however, as Metcalf (1912 b:81–82) says, there seems to be no substantiating evidence for such an assertion. The marked abundance of dead branches on slow-growing chestnuts has been taken as a sign of their debility.

The opinion that the chestnut has not always been thrifty throughout its range is based on observations of various writers. Clinton (1913: 407–408) discusses this subject, quoting extensively from many sources. He states: "It is well known that in times past the chestnut trees in this country have suffered severely in certain districts, particularly in the South, in some cases being practically exterminated, so that their range is now considerably lessened from what it was originally. Strangely enough, no one has surely accounted for any of these devastations. Personally we believe that this tree is extremely susceptible to changes in the natural environment, and that such changes, with water playing an important part, have been the chief factors back of the gradual decline of this important forest tree. Other factors, such as forest fires, deterioration through repeated cuttings, insect and fungus attacks, are contributing causes varying in different localities."

THE DISEASE

NAME

The present epiphytotic disease of the chestnut has become known by several common names. Metcalf (1908 a) applied the name "bark disease," and since that time this name, as well as the name "blight," has been used by most writers. Murrill (1908 b) used the name "canker," but unfortunately this name has found preference with but few writers.

There are arbitrary rules, at least, for the naming of types of diseases. The term "canker" has come to mean, both to the pathologist and to the layman in this country, a disease which causes the death of definite areas of the bark of the limbs or the trunks of trees. The various apple-tree cankers are well known. There is no conflict in meaning, therefore, between the names "bark disease" and "canker," for they are synonymous; but, since we already have in common use the name "canker,"

[8] Zon, Raphael. Chestnut in southern Maryland. U. S. Forestry Bur. Bul. 53:1–31. 1904.

there is no logical reason why we should not use it for this disease. Pantanelli (1911) says this name does not seem very exact, as very different alterations of a tree are understood by the word "canker." In a later article, however, Pantanelli (1912:869) used the name "canker."

It is true that European writers attach to the name "canker" the additional significance of a callus formation around the diseased area. Thus they retain the original Greek meaning of the root of the word, which meant an excrescence on the limbs of trees.

The name "blight" has been more generally applied to rapidly spreading and destructive diseases of herbaceous plants or the herbaceous parts of woody plants. As the term "blight" in the case of this disease signifies only a symptom of the disease, not the lesion, it is evidently not so appropriate as either of the other two names. The authors of this bulletin prefer to use the name "canker," and in order that the name may be entirely specific it should be called the Endothia canker of the chestnut.

HISTORY

In the first published account of the Endothia canker, Merkel (1906:97) says: "This disease was first noticed in the New York Zoological Park, in a few scattered cases which occurred during the summer of 1904." In a letter to the writers Mr. Merkel adds: "No indication of the chestnut-tree disease was noticed by me previous to the year 1904. In 1904, however, toward the latter part of the season, I noticed that certain very old chestnut trees were suffering in certain portions of their tops from some trouble or other, but no investigation was made of the cause. . . . Early during the following year, in fact on June 17, I became thoroughly alarmed and sent to the Bureau of Forestry a specimen of the disease on a young tree and a letter asking for information."

Metcalf and Collins (1909:45) state: "Even at that time [referring to the discovery of Merkel in 1904] it is certain that it had spread over Nassau county and Greater New York, and had found lodgment in the adjacent counties of Connecticut and New Jersey. No earlier observation than this [1904 by Merkel] is recorded, but it is evident that the disease, which would of necessity have made slow advance at first, must have been in this general locality for a number of years in order to have gained such a foothold by 1904. Conspicuous as it is, it is strange that the fungus causing this disease was not observed or collected by any mycologist until May, 1905, when specimens were received from New Jersey by Mrs. F. W. Patterson, the Mycologist of the Bureau of Plant Industry."

This was the starting-point of the numerous investigations conducted by a large number of workers. Metcalf and Collins (1911:5) state:

" There is reliable evidence, however, that it was present on Long Island at least as early as 1893." This statement was based entirely on the recollections of certain observant nurserymen.

ORIGIN OF THE EPIPHYTOTIC

In his first publication Murrill (1906 a: 153) advances the theory that " it is possible that the conspicuous ravages of the disease about New York City are largely due to the severe and prolonged winter of 1903–04, during which many trees of various kinds were killed or injured." Metcalf (1908 a: 56) believed it possible that the Japanese chestnut had been the means of introducing a new fungous disease to this country. Clinton (1909: 887), after discussing observations that he had made, states: " From the preceding account one can readily see that the writer believes that the fungus alone is not entirely responsible for the havoc. . . . . Winter injury in 1903–04, aggravated by the droughts, especially that of 1907, we believe to have been important factors in handicapping the trees so that the way was opened for further serious injury by the fungus."

From these statements and the evidence cited for upholding this view, it is seen that Clinton did not believe a new and dangerous pathogen was being dealt with; but, as he states later, he believed that a native obscure fungous disease had suddenly sprung into prominence, due more to the condition of the host than to the potentialities of the organism. These two quite divergent opinions were each based on circumstantial evidence which will be more fully treated under etiology and ecologic relations. As a solution of the problem pathologists welcomed the finding of the disease in China in 1913, for this furnished a satisfactory basis for explaining many factors concerning the epiphytotic.

It therefore seems certain that Metcalf's assumption as to the origin of the outbreak has been proved correct. The opposing views of others, which no longer have any significance in accounting for the origin of the disease, are nevertheless important points to be considered under the influence of ecologic factors on the fungus, the susceptibility of the host, and the possible augmentation of the epiphytotic.

SPREAD IN THE UNITED STATES

The rapidity of spread has been phenomenal, and the completeness of destruction is without parallel in the annals of plant pathology. Merkel (1906) wrote in November, 1905: " Since that time [1904], however, it has spread to such an extent that to-day it is no exaggeration to say that ninety-eight per cent of all the chestnut trees in the parks of this borough [Bronx] are infected." Metcalf and Collins (1909: 45) state that specimens were received from New Jersey in May, 1905. Murrill (1906 a:

143) states in June, 1906: "The same disease has been found to exist among the chestnuts of New Jersey, Maryland, and Virginia." As to the authenticity of the records in Maryland and Virginia at this date the writers of this bulletin are in doubt.

Murrill (1906 b:203, 207) states in September, 1906: "I now know of very few chestnut trees in this portion [Bronx] of the city that appear to be worth trying to save and I do not consider any immune"; and further: "Mr. Levison reports all the chestnut trees of Forest Park, Brooklyn, to be either dead or dying, and many in Prospect Park to be seriously affected. Wherever he has found the chestnut tree in Brooklyn, he has found the disease."

Metcalf (1908 a:55) states in February, 1908: "The bark disease.... is now reported from Connecticut, Massachusetts, Vermont, New York as far north as Poughkeepsie, New Jersey, Pennsylvania, and possibly Delaware." In the same month Murrill (1908 a:26) reports approximately the same distribution, and adds Maryland to the list. Clinton (1908:345) reports that the disease has become common in the neighborhood of Stamford, Connecticut. Hodson (1908:4–5), after giving more nearly exactly the location of diseased areas in each State, sums up the situation thus: "The range at present, then, includes eight states, Connecticut, New York, New Jersey, Pennsylvania, Delaware, Maryland, Virginia, and Massachusetts." Mickleborough (1909) reports the presence of the disease in six counties in eastern Pennsylvania, and Clinton (1909: 881–885; 1911:716–717; 1913:371–372) gives the data regarding the spread of the disease in Connecticut.

Metcalf and Collins (1909:46) present a map showing the distribution at that time in so far as it was known. By this map it was shown that the disease was found in Rhode Island and southern Connecticut, in New York, New Jersey, and Delaware, and in eastern Pennsylvania, Maryland, and Virginia. They add: "It can be quite confidently stated that the bark disease does not yet occur south of Virginia and at only a few points in that State." Metcalf (1910) states: "At the present time it has spread from Saratoga county, New York, and Suffolk county, Massachusetts, on the north and east, to Bedford county, Virginia, on the south, and Greenbriar and Preston counties, West Virginia, and Westmoreland county, Pennsylvania, on the west."

Metcalf and Collins (1912) publish a detailed map which shows the results of more nearly accurate surveys of the territory along the border line of the spread of the disease. No new States were added to the distribution as already given above, although spot infections were found in many places in Massachusetts, western Pennsylvania, Maryland, and northern Virginia.

The latest published information is given by Metcalf (1914: 8), who reports: "The disease is now generally distributed in native chestnuts from Merrimack county, New Hampshire, and Warren county, New York, on the north, to Albemarle county, Virginia, on the south. In New York the western border of distribution is sharply delimited by an area without chestnut trees—a natural 'immune zone'—which extends southward along the eastern borders of Fulton, Montgomery, and Schoharie counties nearly to the Pennsylvania line in Delaware county. Consequently, in New York the range of the disease is at present practically limited to the valley of the Hudson. In Pennsylvania the western limit of general infection is roughly along a curved line extending from the northwest corner of Susquehanna county to the eastern border of Clearfield county and on to the southwest corner of Fulton county. West of this line the advance infections were cut out by the Pennsylvania Chestnut Tree Blight Commission. The disease has not yet been found in Ohio or Indiana. In general it appears to spread northeastward and southwestward, following the direction of the ridges of the Appalachians, much more rapidly than westward, across the ridges and valleys.

"Scattering infections occur outside of this area. Of these, the outposts are two infections on planted chestnuts in Franklin and Androscoggin counties, Maine, and one infection in a nursery in North Carolina. There is reason to suppose that the North Carolina infection, and an orchard infection in British Columbia, owe their origin to trees imported directly from the Orient."

The data for the map, Fig. 77, were furnished by Dr. Haven Metcalf, and represent the distribution known to him on March 1, 1914.

One might infer from the above that the disease has been spreading in ever-widening circles from the region about New York City. However, Clinton (1913: 374) remarks: "This apparent wave of progress, however, is in part due to a corresponding wave of interest on the part of the people to locate a disease so generally discussed." He, as well as many others, believes that the disease has not started from a single center, but that other affected localities, such as those in Warren county, New York, Bedford county, Virgina, and Lancaster and York counties, Pennsylvania, are nearly as old as that about New York City. Even though the disease was imported from the Orient, as now seems certain to have been the case, it is reasonable to suppose that there were centers of infection started at distant places by the shipment of nursery stock. None of these outlying centers, however, have developed areas of total destruction such as that around New York City; so that it is still reasonable to suppose, from the evidence at hand, that this was the original center and that all other centers were early spot infections originating from it or from later direct importations.

DISTRIBUTION IN NEW YORK STATE

One of the writers of this bulletin (Rankin, 1914) reports the results of the surveys conducted in New York State as follows:

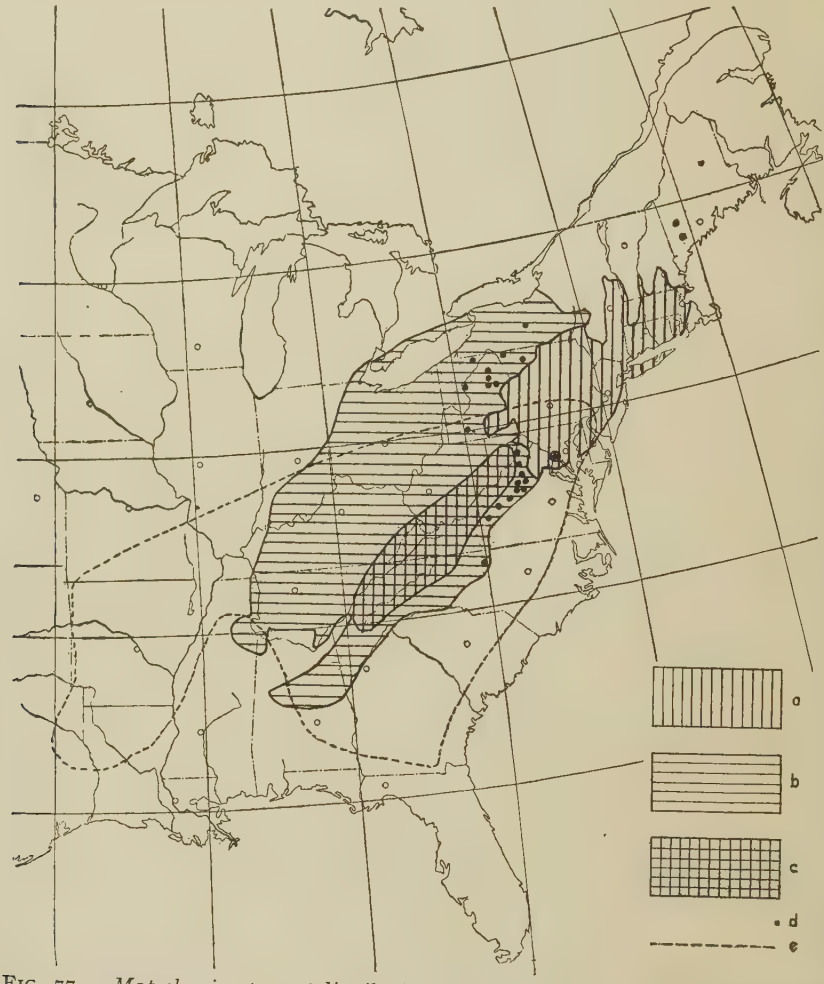

FIG. 77.— *Map showing present distribution of the chestnut canker in the United States. Furnished by Dr. Haven Metcalf*

a, Area over which the chestnut canker is abundantly present

b, Area of natural range of Castanea dentata as yet unaffected except for a few spot infections

c, Area of heaviest stands of chestnut

d, Location of known spot infections

e, Limit of natural range of the chinquapin, Castanea pumila

"At the outset of this investigation [May, 1911] the information available concerning the distribution of the disease attributed the northern limit of its range to southern Dutchess and central Orange counties.

It was known to be generally established in the western part of Long Island, Staten Island, and the counties of New York, Westchester, and Rockland. It was also expected that Orange county had been invaded to some extent."

Range of the chestnut in New York State

Reference to the accompanying map (Fig. 78) shows that the chestnut region of the State lies in two almost separated areas — the Hudson Valley, and the central and western parts of the State — and the two areas are connected by a narrow band of chestnut skirting the Delaware

FIG. 78.— *Map showing distribution of the chestnut canker in New York State*
a, Canker abundantly present in 1911
b, Spot infections numerous in 1911
c, Spot infections numerous in 1913
d, Area containing more or less chestnut that is healthy

River in Sullivan and Delaware counties. There is no chestnut to be found in the Adirondack Mountains, and but little in the Catskill Mountains and the Highlands in Ulster, Sullivan, and Delaware counties. The extreme northeastern corner of the State contains but a few scattering chestnut trees.

Conditions in 1911

The accompanying map (Fig. 78) shows the results of the survey in fixing the limits of spread of the disease at that time. East of the Hudson River the disease was found abundantly as far north as Albany. The chestnut is most abundant along the state line between New York and Connecticut and Massachusetts, in the range of mountains making up

the Berkshires. Along the Hudson River the chestnut is plentiful, but not so abundant as in the hills. It was along the state line, in these heavy stands of chestnut, that the disease had made the greatest ravages. It was present, however, in every locality where there was any quantity of chestnut. Practically every town east of the Hudson River is represented in the list of those in which the disease was found. In those towns in this region from which the disease was not reported, the stand of chestnut was slight. In the central part of Rensselaer county there is little chestnut, and especially is this true of the part near the Hudson River. Along the state line separating New York from Massachusetts and Vermont, in the continuation of the Berkshires northward, there is considerable chestnut, but no disease was found there.

The natural northern limit of the chestnut, as is seen by the map, is in the towns of Fort Ann and Granville, in Washington county. The disease was found very abundant throughout Washington county. Many lumbermen were cutting chestnut as rapidly as possible in the towns of Hartford, Argyle, and Hebron, in order to keep ahead of the disease.

West of the Hudson River the disease had not advanced so far north. Practically the entire stand of chestnut in Orange county and in southern Ulster county was diseased. In the Catskill Mountains proper there is much chestnut in the valleys opening into the Hudson River Valley. In the southernmost of these valleys the disease had spread as far as chestnut is to be found, but in the Esopus Valley and in the valleys north of it the disease had spread for only a little distance. In Albany county the chestnut is scattering, except in the Helderberg Mountains, and it is very scarce in the western part of the county. A complete survey of Albany county was not made, owing to the unimportance of this information; however, the disease was found general in the southern part of the county along the Hudson River.

The connecting link between the chestnut region of the Hudson River Valley and that of the central and western part of the State is the strip in Sullivan county along the Delaware River. Chestnut is abundant here for fifteen to twenty miles away from the river. Many isolated spot infections were found here. The general spread extended about as far up the Delaware River as Port Jervis. A few spot infections were found in southern Delaware county along the Delaware and Susquehanna rivers. One spot infection was found at Masonville, Delaware county, and was destroyed. No infections were known west of this region in 1911. A special investigation of the cause of the dying of the chestnut near Cooperstown was made, but it was found not to be due to the canker disease.

Conditions in 1913

A hasty resurvey of the territory bordering on the line of advance determined in 1911 was made in September, 1913, for Doctor Metcalf. The area just west of the chestnut-free belt was covered, and the line as drawn on the map (Fig. 78) was fixed as representing approximately the limit of spot infections at that time. The strip along the Delaware River where isolated spots were found in 1911, was found at this time to be abundantly affected. The disease seems not to be spreading so rapidly north as west, and no infections were found in Otsego county just west of the chestnut-free belt in the Catskills. The width of this belt varies from thirty to forty miles, and it is interesting to note that apparently this distance has furnished a natural barrier which has so far impeded the dissemination of the fungus westward from the diseased areas along the west bank of the Hudson River.

ECONOMIC IMPORTANCE

Several writers have attempted to estimate in pecuniary value the losses due to the Endothia canker. The disease not only kills the merchantable trees on which a pecuniary value can be placed, but also destroys the young growth which would have been cut in the future, the value of which cannot be determined accurately. Aside from this, the æsthetic loss from the destruction of thousands of shade and ornamental trees cannot be computed in dollars.

Great as the damage has been, it seems that estimates are worth but little because of the lack of accurate data. Clinton (1913:379) tersely sums up the situation: "Just how this loss is estimated is not made very clear. To the writer it seems to be largely guess work. However, it is interesting to note these figures in order to compare them with losses given for other fungous diseases and insects." Murrill (1908 b:111) states: "The amount of damage done by it, in and about New York City, where it has been most carefully observed, probably reaches a total of between five and ten million dollars." Metcalf and Collins (1911:5) regard $25,000,000 as a conservative estimate of the financial loss from this disease up to 1911. Metcalf (1913:364) states: "The estimate of $25,000,000 made in 1911 as representing the loss up to that time was probably much too conservative. But the total loss to date is insignificant compared with the loss which will ensue if the disease once attacks the fine chestnut timber of the South Appalachians."

SYMPTOMS

With few exceptions every writer on the subject has described the symptoms of the disease, and many writers have published excellent

illustrations. The effects of the disease on the general appearance of the tree are most noticeable during the summer, when the trees are in leaf. In regions where the disease is common, the newly affected limbs and twigs are girdled in large numbers during the summer, and the brown, shriveled leaves are readily seen even at a distance. This most striking symptom is common during July and August, while the healthy parts of the tree are still green.

FIG. 79. — *Tree during winter, showing the leaves still clinging to the branches, which have been killed by girdling cankers*

The dead leaves also remain clinging to the limbs during the winter (Fig. 79). If the girdling of a limb is completed during the time when the burs are maturing, the latter often remain on the branches over winter. If, however, the girdling takes place after leaves and burs are shed and before the leaves come out in the spring, the leaves do not attain their full size and are pale and distorted. This is a common symptom in May and June (Fig. 80). Dead limbs without attached leaves or burs are often indications of the presence of the canker disease (Fig. 81). Water sprouts or suckers are commonly developed just below the cankers. Thick clumps of suckers on the trunk (Fig. 82), on the branches, or at the base of the tree, are often the only telltale evidence of a developing canker. Such growths

are very conspicuous the year round. In time they are killed either by the canker's invading the area from which they grow or by the infection of the suckers themselves.

FIG. 80.— *A diseased and a healthy tree in midsummer. Notice the poorly developed leaves on the diseased tree (at the left)*

Cankers on smooth bark are especially conspicuous (Plate XXXVI, frontispiece). They can be seen for a long distance because of their reddish tinge in contrast to the healthy green bark. The cankers are either sunken or swollen diseased areas of the bark (Plate XXXVII). They occur on branches of all sizes — only rarely, however, on first-year twigs. The usual shape of the canker is ellipsoidal, being longer

FIG. 81. — *Chestnut woodland devastated by the canker. Photograph taken in midsummer*

in the direction of the long axis of the limb. The margin of the canker is usually fairly regular, but it may be irregular.

Metcalf (1914) says the cankers formed on the Chinese chestnut become deeper year by year as healthy wood is formed about them, thus causing a condition similar to the European apple-tree canker.

Usually about a month after inoculation the bark of the affected area becomes covered with numerous small pimples. Under moist conditions, following rains, long, twisted, yellow tendrils are extruded from the

TYPES OF CANKERS ON CHESTNUT CAUSED BY ENDOTHIA PARASITICA

FIG. 1.—*Part of canker showing twisted fycnospore tendrils*

FIG. 2.—*Stromata on smooth bark, showing papillae, at the tip of which are the orifices of the perithecia*

FIG. 3.—*Lines of pycnospore tendrils in crevices of rough bark*

EXTERNAL APPEARANCE OF FRUITING STAGES

FANS OF MYCELIUM IN THE CAMBIUM AREA AFTER THE BARK HAS BEEN PEELED OFF

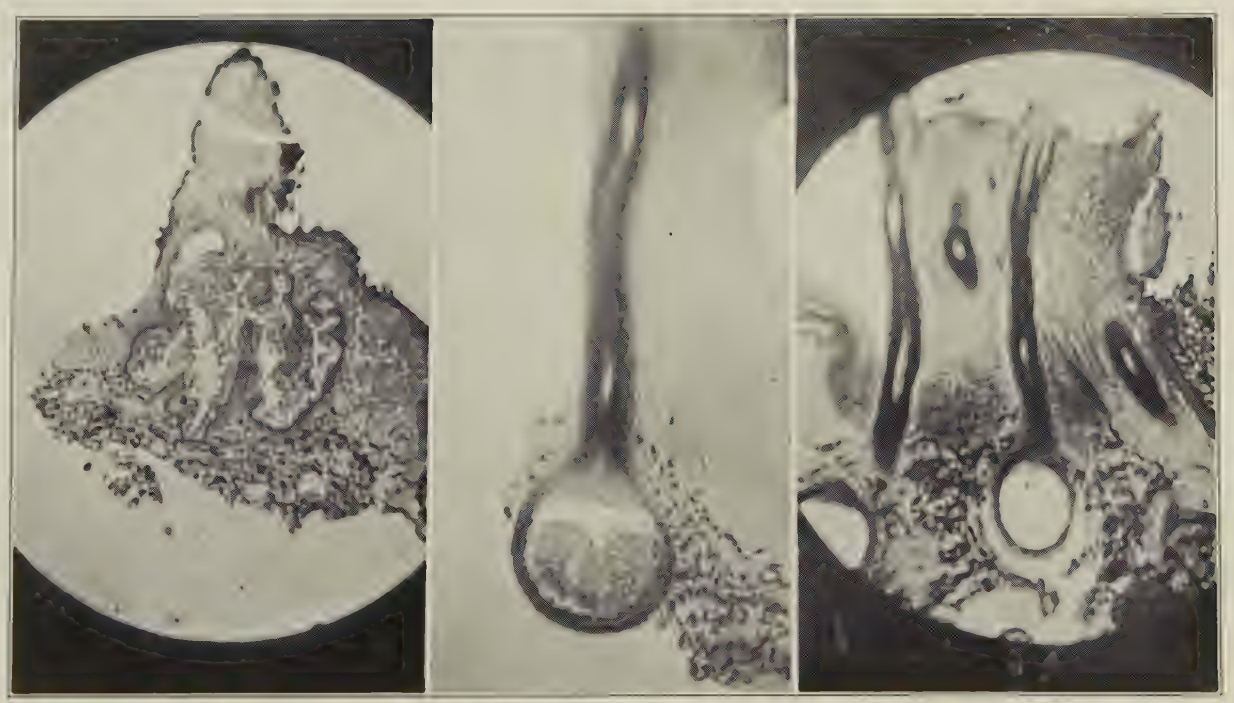

FIG. 1.—*Section of stroma showing mature pycnidium of irregular shape. Pycnidium partly filled with spores*

FIG. 2.—*Longisection of a perithecium partly filled with asci*

FIG. 3.—*Section of stroma showing two empty perithecia with their black necks extending to the ostioles at the surface*

LONGISECTIONS OF STROMATA SHOWING FRUIT BODIES

ruptured apices of these blisters (Plate XXXVIII, Fig. 1). Older and larger cankers have the pimples and the yellow tendrils confined to the margin, while nearer the center reddish-brown pustules have been pushed out through the outer bark. These pustules, when fully developed, measure one sixteenth of an inch or more in diameter and have numerous papillæ on the upper surface, each with a black dot at the tip (Plate XXXVIII, Fig. 2). Cankers on rapidly growing limbs are usually outlined by a distinct ridge of slightly hypertrophied tissue. Where the whole cankered area is hypertrophied the bark usually splits longitudinally (Plate XXXVII). As the canker becomes older the bark splits and cracks, and after a few years it breaks away entirely and leaves the wood bare (Fig. 83). When the tree is thoroughly diseased — and this may take only two or three years after the first infections occur if they are at the top of the tree — the brown pustules cover the bark of the trunk and the branches, giving the tree the distinc-

FIG. 82.—*Water sprouts on the trunks, indicating cankers just above them*

tive brown hue. When the fungus affects rough bark, no shrinking nor swelling nor change in color is evident, and it is only when the yellow tendrils, or later the brown pustules, are produced in the crevices of the bark that an outward indication of the diseased area appears (Plate XXXVIII, Fig. 3).

FIG. 83.— *Tree that has been dead for several years, showing the bark falling off in patches and exposing the wood*

By examining the character of the destruction of the bark one may determine other diagnostic characters. When the bark is peeled from the edge of a canker it is found to be flaked with tawny, fan-shaped areas, which, once seen, are easily recognized (Plate XXXIX). The invaded tissue is changed to a light sienna brown and appears in marked contrast to the normal light-colored bark. Thick bark that is affected is reduced to a mass of shreds, which are a uniform dark brown in color. The first layers of wood and the medullary rays also turn brown under the cankered area.

ETIOLOGY

Name of the causal organism

It has been proved by Murrill (1906, a, b, and c) that the Endothia canker of chestnut is produced by a fungus which he refers to the genus Diaporthe, of the Sphaeriales. Believing that it had not been previously described he gave it a new specific name, *parasitica*, recalling its destructive parasitic habit.

Rehm (1907) declares that it belongs among the Hypocreales in the genus Valsonectria, and proposes the combination *Valsonectria parasitica* (Murr.) Rehm.

Clinton (1909:888) quotes Farlow as stating that "it comes more naturally under the genus Endothia, and is closely related to *E. gyrosa*." The latter is a saprophytic species which, as limited by Saccardo, was first described from North Carolina by Schweinitz in 1822 as *Sphaeria*

gyrosa on Fagus and Juglans, and in 1828 by Fries as *S. radicalis* on Fagus (or Quercus) from the same State, and which is found commonly on oaks, chestnuts, and a number of other trees in Europe as well as in America.

Von Höhnel (1909) says that *Diaporthe parasitica* Murr. is identical with *Endothia gyrosa* (Schw.) Fückel; also that the genus Valsonectria is not different from Endothia, which he would place among the Hypocreales because of its bright-colored stroma rather than in the Sphaeriales where it is usually placed. He gives two reasons why the fungus is an Endothia rather than a Diaporthe: (1) the bright-colored stroma is typical of the former, but not of the latter; (2) the conidial fruit form of Diaporthe is Plenodomus Preuss (Phomopsis Sacc.), while that of Endothia is Endothiella Sacc., characterized by irregular chambers in the stromata, without a definite wall and with small, rod-shaped spermatia.

Clinton (1912 c:79–80), at the Harrisburg Conference in February, 1912, states that he believes *Diaporthe parasitica* to be closely related to *Endothia gyrosa*, but of a different species. In a footnote, written after the conference, he states that he found *E. gyrosa* common as a languishing parasite or saprophyte on chestnuts and oaks in Virginia, North Carolina, and Tennessee; and that it differs from *Diaporthe parasitica* in that the stromata are less luxuriant, and especially that the ascospores are smaller and narrower, but he is not sure " whether these differences are those of a strain, variety, or distinct species." At the same time Farlow (1912 a:74) gives it as his opinion that *Endothia gyrosa* and *Diaporthe parasitica* are identical. Rankin (1912 b:47) states that he found a saprophyte common on the chestnuts in Virginia which is indistinguishable from the canker fungus. So far as the literature shows, Rankin was the first to find this saprophytic Endothia on chestnuts in America.

All the writers quoted above consider *Endothia gyrosa* (Schw.) Fr. and *E. radicalis* (Schw.) de Not. as synonymous. Shear (1912 a) questions their identity, and in the same paper states that there is a specific difference between *Diaporthe parasitica* and *Endothia radicalis* (Schw.). It appears from Shear's later papers (1912 b and 1913 a) that he was applying the latter name to a form with bacilloid spores found commonly in the Southern States, first brought to notice by Farlow (1912 a:74) and later called by Clinton (1912 a) the " linear-spored Endothia."

Farlow (1912 b) in Science repeats his previous statement that *Diaporthe parasitica* and *Endothia radicalis* (European form) are identical, and states that his opinion is shared by von Höhnel and Saccardo.

Since 1909 the generic position of the fungus has not been seriously questioned, all authorities apparently agreeing that it is an Endothia. Outside those already mentioned, the most valid reason for placing the fungus in Endothia rather than in Diaporthe or Valsonectria may be

stated thus: Endothia was erected in 1846, with *Sphaeria gyrosa* Schw. as the type. When the new fungus that causes the canker was studied, it was found to resemble *E. gyrosa* (as defined by Saccardo) so closely that even such authorities as Saccardo, von Höhnel, and Farlow could not distinguish the two species. Also the very few other species that have been referred to this genus resemble these two so closely that no one would think of putting them in different genera. Wherever one is placed they must all go. If one is placed in Diaporthe they must all be placed there, that is, the whole genus must be combined with Diaporthe. But, according to our rules of nomenclature, when the members of two genera are combined they take the name of the species first described, which in this case happens to be an Endothia. Therefore, even if there were not sufficient generic differences to keep the two genera separate, the canker fungus would still be Endothia, not Diaporthe nor Valsonectria — genera which were established many years later. The question from this time on was in regard to the specific name to be applied.

Shear (1912 b), after a summer in Europe, stated that the *Endothia radicalis* of European authors is morphologically identical with *Diaporthe parasitica;* also that the American *Endothia radicalis* (Schw.) is the long-spored form of the Southern States; also that the latter is different from the European *E. radicalis*. He later retracted the first and the second of these statements (1913 a).

Anderson and Anderson (1912 a) made a study of the saprophytic American Endothia, which they found common on chestnuts and oaks in southwestern Pennsylvania, Virginia, West Virginia, and Tennessee. They found marked morphological, biological, and cultural differences between this and the true canker fungus. They consider these differences of specific value, and propose the new combination *Endothia parasitica* (Murr.) for the canker fungus and *E. virginiana* sp. nov. for the saprophytic form. The most noticeable morphological differences are:

E. parasitica	*E. virginiana*
Mycelium forms thick, fan-like mats in the bark and on the cambium	No such mats perceptible
Size of ascospores, 8.6 by 4.5 μ	Size of ascospores, 7 by 2.98 μ
Shape of ascospores, broad: proportion of diameter to length, 1:2.7	Shape of ascospores, narrow; proportion of diameter to length, 1:1.9
Constriction at septum of ascospores distinct	Constriction very slight, if any
Length of ascus, 51.3 μ	Length of ascus, 34 μ

Biologically, the former is a virulent parasite and the latter a saprophyte. Cultural differences were found on every artificial medium on which the two were grown in pure culture. The reasons given by Anderson and Anderson for creating a new specific name for the saprophyte are: (1) Shear had identified the long-spored southern form as *E. radicalis* and this was entirely different from the species under consideration; (2) an examination of the type material of *E. gyrosa* (Schw.) Fr. in the Schweinitzian herbarium at the Philadelphia Academy of Science, and a comparison with the original description and all the early literature on this fungus, convinced them that it was a form entirely different from any species of Endothia now known; (3) there was no other described species of Endothia besides the above two which closely resembled the fungus that they were studying in western Pennsylvania.

Clinton also studied the relationships of these two forms and found practically the same differences as did the Andersons, besides additional cultural differences; but he considers the differences varietal, not specific. Shortly after the appearance of the paper mentioned in the preceding paragraph, Clinton (1912, a and b) published two papers in which he states that the European Endothia and the American *E. virginiana* Anders. are identical with *E. gyrosa* (Schw.) Fr. He proposes *Endothia gyrosa* var. *parasitica* as the proper name for the canker fungus. He emphasizes especially the difference in the shape of the ascospores, and calls the species the " narrowly-oval-spored " and the variety the " broadly-oval-spored " Endothia.

At about the same time, Pantanelli (1912), in Europe, studied the relationship of the European Endothia to the American canker fungus, and found important morphological, biological, and cultural differences which he considers of specific value; he designates the latter form as *E. parasitica* (Murr.) Anders., and the European form as *E. radicalis* (Schw.) Fr.

Shear (1913 b) examined asci and ascospores of the type specimen of *E. radicalis* (Schw.) from the Fries herbarium at Upsala, Sweden, and found them to correspond with those of *E. virginiana* Anders. and the form discussed by Clinton as *E. gyrosa.*

Shear and Stevens (1913 a) were the last to study in detail the relationships of the species of Endothia. They studied especially the cultural characters, and on nearly every kind of medium tried they found characters by which *E. parasitica* can be distinguished from the other species. They give as the correct names of the species here under consideration:

1. *Endothia parasitica* (Murr.) Anders., the true canker fungus
2. *Endothia radicalis* (Schw.) de Not., the saprophyte found first on chestnuts in this country by Rankin, later called *E. virginiana* by

the Andersons (1912, a and b), and discussed under *E. gyrosa* by Clinton (1912, a and b) as the "narrowly-oval-spored Endothia"

3. *E. gyrosa* (Schw.) Fr., the bacilloid-spored southern form, which Shear (1912 b and 1913 a) had previously called *E. radicalis* (Schw.) and Clinton had discussed under the name of *E. radicalis* (Schw.) Farl., or the "linear-spored Endothia"

The last of the three need not be considered especially at this time, since it has not been confused with the true canker fungus. Finding no intermediate forms between the first two, Shear and Stevens conclude that "unless such intergrading forms should be discovered later, there seems to be no way of escaping the conclusion that the two organisms are specifically distinct, according to the most conservative taxonomic standard of species prevailing at present in mycology."

In this publication the canker fungus will be referred to as *Endothia parasitica*. The complete synonymy is as follows:

Endothia parasitica (Murr.) Anders. Phytopath. **2**:210, 262. December, 1912

Diaporthe parasitica Murrill. Torreya **6**:189. 1906

Valsonectria parasitica (Murr.) Rehm. Ann. myc. **5**:210. 1907. Ascom. exs., fasc. 39, no. 1710

Endothia gyrosa var. *parasitica* (Murr.) Clinton. Science **36**:913. December, 1912

Concerning the pycnidial stage of the fungus, it was first referred to the genus Cytospora by Patterson (Clinton 1909:879), and shortly afterward by Clinton (1909) to the same genus. Since that time it has frequently been referred to in the literature under that name. Reasons for considering this disposition of it as incorrect will be given in the discussion of morphology and life history. In 1906 Saccardo (Ann. myc. **4**:272) erected the genus Endothiella, based on the pycnidial form of *Endothia gyrosa* (*E. radicalis* [Schw.] de Not., according to Shear). Since the imperfect stages of our American Endothiae are practically indistinguishable, it is evident that the pycnidial form of *E. parasitica* should be referred to that genus, if there is ever any need of considering it apart from the perfect stage.

History of the pathogen

When the disease was first noticed in this country in 1904 it was a comparatively simple matter to determine the association of the fungus with it. But where did this fungus come from, and why had it not been noticed before? There were only two possibilities: (1) either the fungus had always been here, but was now for the first time brought to notice; or

(2) it was an introduced species. When a fungus produces a sudden and destructive epiphytotic the presumption is that it has been introduced from some foreign country — a presumption that is supported by numerous well-known analogous cases in the past. But, on an investigation of conditions in other countries, no record of a similar disease of the chestnut could be found. This, however, does not invalidate the importation theory, since it is a fact well-known to plant pathologists that a fungus may be an inconspicuous and comparatively harmless parasite in its native country and yet may produce a destructive epiphytotic on being taken to another country.

This theory was first advanced by Metcalf (1908 a). Since he was unable to find any record of the occurrence of the disease in this country, and since he found the Japanese chestnuts more or less immune, he suggested that the fungus was a native of Japan and had been introduced here on imported trees. The presumption would be that, spreading to our native chestnuts, the fungus found these less resistant and more favorable to its growth; hence its rapid spread throughout the eastern States. In February, 1912, just previous to the Harrisburg Conference, Metcalf (1912 b:77–78) discussed his theory in part as follows:

"My own working hypothesis is, that the parasite is an importation from some country other than North America. The resistance of the Japanese and Korean chestnuts, coupled with the fact that the Japanese chestnut has been extensively imported and grown in that part of the country whence the disease appears to have spread, suggests that Eastern Asia may be the home of this parasite. . . . That the parasite has come from Europe seems less probable, in view of the fact that, according to Pantanelli, as well as according to my own inoculations under greenhouse conditions, the European chestnuts show no resistance to the disease. . . .

"The main fact in support of a foreign origin of the disease is its unquestionable spread in all directions from the vicinity of New York City. It is further suggestive that the oldest centers of infection located outside the vicinity of New York City — Bedford county, Virginia, and Baltimore county, Maryland — contained chestnut orchards with Japanese chestnut trees, possibly also European varieties. If *Diaporthe parasitica* is a native fungus, or has evolved from a native saprophyte, it is necessary to assume that the saprophyte was very limited in range, or that the evolution to a condition of parasitism occurred in only one, or at most a very few localities, or that there is a chronological sequence in its evolution proportional to its distance from New York City. Any of these assumptions are a severe tax on the scientific imagination."

Clinton (1909, 1911, 1912 a, 1912 c, 1913) holds to the opposite view;

that is, that the fungus has always been in this country as an inconspicuous saprophyte or a weak parasite, and that it became virulent about 1904 because the trees were in a weakened condition due to winter injuries and unfavorable weather conditions, as well as faulty silvicultural methods such as continuous coppicing. He briefly sums up his reasons for his theory as follows (1913:416–417):

"The writer's reasons for believing the chestnut blight is native to this country may be summarized as follows: (1) It has never been found in any other country. (2) It is very closely related to *Endothia gyrosa*, apparently developing from it as a distinct variety, and this species is a native fungus in this country as well as in Europe. (3) The limits of distribution of *E. gyrosa* and the chestnut blight overlap at least in the region covered by Washington, D. C., to southern Pennsylvania, while *E. gyrosa* occurs south of this common area and the chestnut blight north of it. (4) We have previously had serious troubles of chestnut trees in this country, and there seems to have been a continued northward movement of these, culminating in the recent trouble in the northern limit. While the chestnut blight has been definitely connected only with this last trouble, the previous ones have never been really explained. (5) The suddenness, et cetera, of the recent blight outbreak has been adequately explained by the writer through the unusual environmental conditions that have weakened the chestnuts in the general regions where the outbreak has occurred. (6) The fact that the chestnut blight fungus was never reported before this outbreak is no more difficult to explain than the fact that *E. gyrosa* had never been reported on chestnut in this country until by the writer a year ago, and yet this is a native fungus widely distributed on chestnut in the South, and has been known there on other hosts since 1822, when described by Schweinitz. They both were, in fact, merely overlooked on the chestnut. (7) Our cultures of *E. gyrosa* vary more from their normal type than do those of the variety *parasitica*, and some of these have varied somewhat toward the variety *parasitica* type. This, however, may have been due in part to bacterial contamination, et cetera."

The strong point in Clinton's argument, and the missing link in Metcalf's, was that the fungus could not be found in eastern Asia. But this link was supplied during the last year, when Fairchild (1913) reported the finding of the fungus in northern China by Meyer, an agricultural explorer of the Office of Foreign Seed and Plant Introduction of the United States Department of Agriculture. As had been expected, the fungus was found to be only weakly parasitic in that country, judging from Meyer's letter, which is quoted here only in part:

"The blight does not by far do as much damage to Chinese chestnut

trees as to the American ones. Not a single tree could be found which had been killed entirely by this disease, although there might have been such trees which had been removed by the ever active and economic Chinese farmers. Dead limbs, however, were often seen and many a saw wound showed where limbs had been removed. . . . The wounds on the bigger majority of the trees were in the process of healing."

Shear and Stevens (1913 b) made cultures from the Chinese specimens and found that all the cultural characters are identical with those of *Endothia parasitica*. Spore measurements agreed very closely, and inoculations made on native American chestnuts produced typical cankers. The writers of this bulletin have grown the Chinese fungus in culture and cannot distinguish it from the American chestnut-canker fungus.

It may now be regarded as practically certain that the early home of *Endothia parasitica* was the Orient.

Morphology

Stromata

Cankers of a season's growth or older show numerous orange-colored or reddish-brown, erumpent and projecting, stromata (Plate XXXVII). On smooth bark the stromata are usually elongated horizontally and average about 2.4 by 1.2 millimeters, by 1.3 millimeter in depth. The part beneath the ruptured cork layer is flattened out on the collenchyma and is broader than the exposed part (Figs. 84 and 85). The stromata, however, vary widely in size with environment and season; they become much larger in moist situations than in dry surroundings where they are exposed to desiccation. On old, rough bark they do not occur singly, as shown in Plate XXXVII, but are found only in the crevices of the bark, often united in solid lines several inches long so that they apparently form one long stroma.

The color varies with age, being sulfur yellow at first, later becoming orange, reddish-brown, and finally cinnamon-brown on the surface, but always lighter-colored on the inside. When in a shaded and moist location — as, for example, on the underside of a log — the stromata remain light yellow.

In the fresh condition the stromata are soft, dry, subcoriaceous, easily torn apart, and of rather loose, indefinite outline. A cross section shows that the center of the stroma is composed of a comparatively loose tangle of branched and septate hyphæ, containing yellow pigment. Throughout the basal parts are scattered stone cells, bast fibers, and remnants of the walls of collenchyma cells. The entire exposed surface of the stroma is covered by a rind layer, in which the hyphal cells are shorter and thicker, almost or quite isodiametric, and with heavier walls than in the interior.

FIG. 84.— *Cross section (diagrammatic) of a mature pycnidium under the cork layer; ostiole not shown. After Heald*

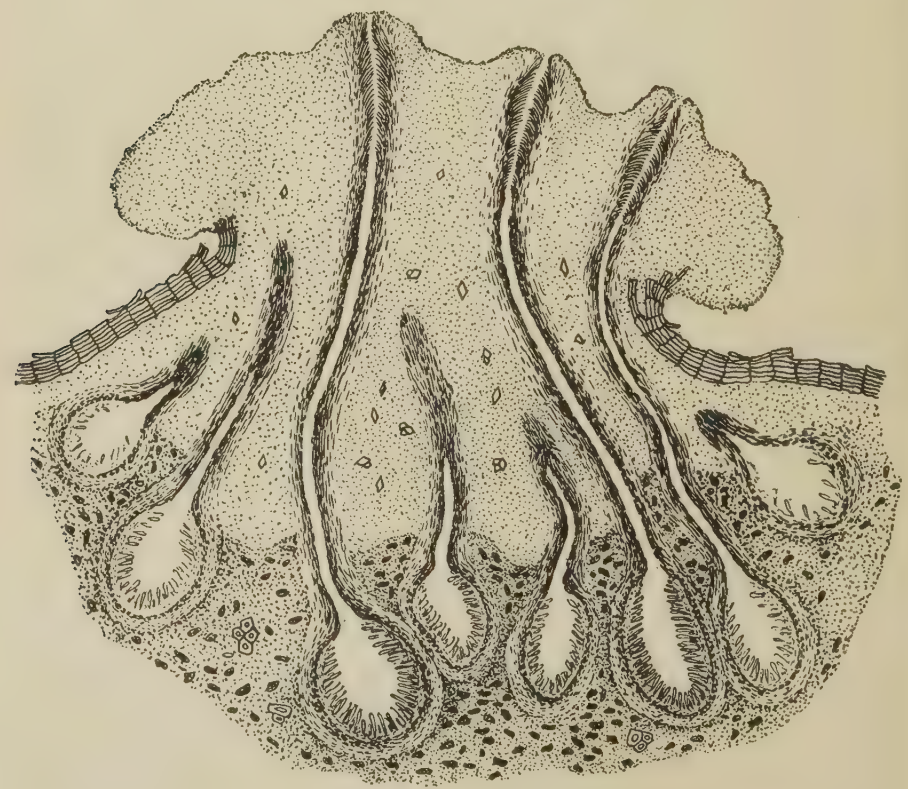

FIG. 85.— *Cross section (diagrammatic) of a stroma, showing mature perithecia. After Heald*

They are closely crowded together, so that in cross section they appear to make up a pseudoparenchymatous tissue. These cells are more densely filled with protoplasm, and contain more pigment, than the interior cells.

Pycnidia

On smooth-bark, young cankers, especially in the summer, the outer cork layer is raised in numerous little blisters, with slender, yellow, waxy tendrils curling from their ruptured apices (Plate XXXVIII, Fig. 1). Under each blister is a single somewhat globose pycnidium, surrounded by a scanty, loose growth of white or slightly yellowish mycelium. There is as yet no definite stroma. The wall of the pycnidium is composed of closely tangled hyphæ; that is, it is not a definite pseudoparenchymatous wall. The cavity may be a fourth of a millimeter in diameter and is almost round in cross section at first, becoming irregular only with age. The conidiophores form a dense, brush-like fringe and extend directly out into the cavity from every point of the wall (Fig. 86). They are of uneven lengths, the majority being 20 to 40 μ long, and are about 1.5 μ in diameter. They may be simple or branched. Spores are cut off successively from the conidiophores or their branches and soon fill the cavity, but, since the production of spores does not cease when the cavity is filled, they are forced out through an irregular ostiole at the top in yellow tendrils. These tendrils take on a reddish tinge as they become old. They vary in thickness from the diameter of a hair to half a millimeter, and in length from a millimeter to three or four centimeters. They occur singly and are usually spirally twisted into one or more coils (Plate XXXVIII, Fig. 1).

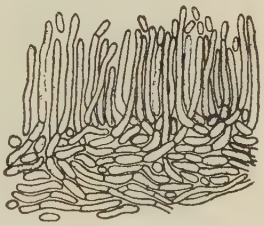

FIG. 86.— *Section of the wall of a very young pycnidium, showing the conidiophores, with the manner of their origin from the hyphæ. Conidiophores become branched in older pycnidia*

The older pycnidia contained in mature stromata differ from these in some respects. The cavity is convoluted or labyrinthiform, and irregular (Fig. 84, and Plate XL, Fig. 1). When cross sections of these stromata are cut, a single section usually shows a number of cavities which do not appear to be connected; but if the entire stroma is cut into serial sections it is found to contain but a single pycnidium with a number of communicating chambers. Only rarely have the writers found stromata containing more than one pycnidium. This stage of the fungus has been referred to the genus Cytospora, on the erroneous idea of a stroma containing many pycnidia. The cavity of the labyrinthiform type of pycnidium often becomes as much as a milli-

meter in diameter. The spore horns from this type are also usually much stouter than those from the former type, and on the bark of old trees, where they arise from lines of confluent stromata in the crevices, a whole line of them may be united in a comb-like manner. The spore horns of both types are usually flat or irregular in cross section. This accounts largely for the way in which they curl. When dry they are hard and brittle and not easily detached or broken.

Another form of pycnidia occurs on cut ends of stumps and logs, and also on both the wood and the inside of the bark where the latter has broken loose. These are superficial, single pycnidia (Fig. 87). A favorite place for them is on the inside of the bark where it has drawn away from the stump around the top, after the tree has been cut. Also, when a log or a stump on which there was a canker is peeled, these pycnidia develop on the surface very quickly. Their production is largely dependent on the water supply; this is illustrated by the fact that in dry weather they develop on the lower side of a log lying on the ground, but not on the upper side. In moist, shaded places they are long pear-shaped or conical, as shown in Fig. 87, or the base may be flattened out slightly on the substratum; but on tops of stumps — where they occur abundantly on the outermost four or five annual rings and where the supply of moisture is not constant — they are flattened out on the substratum and do not stand out free as shown in the figure, and they also have more of a tendency to run together. In color they are deeper red than the stromata, but have light yellow, conspicuous, beak-like ostioles. They are surrounded by no stroma whatever and stand out free except as stated above. They measure about a quarter of a millimeter in diameter and the same in height. The outer wall is perfectly smooth as seen under a hand lens. Often several pycnidia unite, but their ostioles remain distinct and give the appearance of a single pycnidium with several ostioles.

FIG. 87.— *Superficial pycnidia on wood, developed in a moist situation*

Pycnospores

The yellow tendrils are composed entirely of small, hyaline spores that are held together by a sticky substance the nature of which has not been carefully investigated. When the tendril is placed in water, it first swells considerably, then the binding substance dissolves and the spores float away free from one another. The spores average about 1.28 by 3.56 μ in size, and are oblong or cylindrical with rounded ends, or slightly oval, usually straight but sometimes slightly curved. The fact that they

are not typically curved is an additional reason why this stage should not be referred to Cytospora. The spore membrane is thin and smooth. The spores are filled with dense homogeneous protoplasm, and each spore contains a single small, elongated nucleus near the center. There is also a polar body in each end.

Perithecia

The mature stromata on older cankers have numerous projecting papillæ on the surface (Plate XXXVIII, Fig. 2). The black speck at the tip of each papilla is the opening of a perithecium, the body of which is located down in the bottom of the stroma and is connected with the apex by a long, black neck. These papillæ may scarcely project above the surface of the stroma, or, on the other hand, they may be a

FIG. 88.— *Stages in the development of the perithecium*

a, Cross section at the stage when the cavity is occupied by young paraphyses. The neck is not shown

b, Tip of the neck in cross section as it appears, pushing up through the stroma at the same stage as shown in (a)

c, Lower part of neck and canal when the perithecium is almost mature, showing periphyses projecting into the canal

millimeter or more in length. They are longer in moist, shaded places than in dry, lighter surroundings. There are commonly fifteen to thirty perithecia in a stroma, but the number varies greatly, over forty having been counted in some cases. In Fig. 85 (page 558) and in Plate XL, Figs. 2 and 3, perithecia in longisection are shown. As seen under the hand lens the wall of the body of a perithecium is gray or lead-colored, while the neck is jet black and shining like anthracite. The mature perithecia measure about 350 to 400 μ in diameter and are mostly spherical, but the shape is often modified by pressure of surrounding perithecia.

Since the perithecia are always in the bottom of the stroma next to the host tissue, the length of the neck varies with the luxuriance of the stroma and the length of the papilla; but, in general, it is four to six times the diameter of the body. The black wall of the neck is composed of densely

interwoven, septate, heavy-walled hyphæ running longitudinally with the long axis of the neck.

Branches of the same hyphæ project out free into the canal and upward toward the tip (Fig. 88, b). They are thin-walled, and react toward stains in a manner different from that of the other tissues. They are especially prominent at the tip of the neck. They are the periphyses.

In cross section the wall of the body is seen to be composed of ten or twelve layers of flattened, heavy-walled cells, very compact and pseudoparenchymatous (Fig. 88, a). Inside this there is a region of two or three layers of thin-walled cells, from the inner surface of the basal part of which the asci grow out into the cavity.

Asci

When the perithecium has become mature the entire cavity is filled with asci — the older, mature ones in the center, and younger ones around the walls. Mature asci are shown in Fig. 89, b, c, and d. They are broadly clavate or oblong, and each ascus contains eight spores in a matrix of epiplasm. The average size of one hundred and fifty asci measured by the writers was 51.2 by 8.9 μ. The arrangement of the spores is irregularly uniseriate or sub-biseriate; but there is little uniformity in this arrangement, since two asci can hardly be found in which the spores are oriented alike. The wall of the ascus is delicate and hyaline, and for this reason is hard to make out in its entirety in unstained mounts. There is a thickened ring about the upper end of the lumen of the ascus which is prominent and shows peculiar staining reactions. When the ascus is lying flat on the slide, the ring appears in optical section as two highly refractive disks (Fig. 89, c).

FIG. 89.— *Ascospores and asci*
a, Three ascospores showing nuclei
b, Ascus with spores when dry
c and d, Asci after swelling in water

Ascospores

The ascospores (Fig. 89, a) are oblong to oval with rounded or blunt-pointed ends, two-celled, constricted at the septa when mature, and on the average about 4.5 by 8.6 μ in size. The walls are thicker than those of the conidia; the septum is distinct and is composed of the same material as the walls. The spores are filled with dense homogeneous protoplasm;

only occasionally have anything like oil globules or vacuoles been seen. Each cell of a spore contains two to four nuclei. Like the conidia, the ascospores have a sticky coating on the outside.

Mycelium

The individual hyphæ are branched, septate threads, the branching being always monopodial, and usually not more than one branch is produced from a single cell. The hyphæ are not of uniform diameter, but vary from 1.5 to 12 μ, and the cells vary from 20 to 50 μ in length. Each cell contains several small nuclei. On most culture media the mycelium becomes yellow after a few days, due to the production of a pigment. The pigment is soluble in alcohol and alkalies, and insoluble in acids. In alkaline solutions the pigment becomes purple. It is a chemical compound belonging to the group known as aurines.

A character of this species which distinguishes it from all other related fungi is the presence in the diseased bark and in the cambium of fan-like mats of mycelium (Plate XXXIX). Each mat consists of a number of bundles of parallel hyphæ diverging from a single point like the rays of a fan. They are flat because they have to squeeze between the bast-fiber zones. The edges of the fans are fairly regular and are surrounded by a darker gelatinous band of disintegrating host cells. The fans vary in length from a millimeter to two or three centimeters. The young ones on the advancing edge are pure white, but the older ones become light yellow or buff. The hyphæ composing each ray branch only sparsely, and are more uniform in diameter than those in agar culture. They do not anastomose in any way.

Pathogenicity

On Castanea dentata

Murrill (1906 a) proved that *Endothia parasitica* is pathogenic on the American chestnut. This fact is so easy of demonstration that it has never been questioned since that time. Constant association of the fungus with the canker is readily noticed by all investigators. The fungus has been isolated and grown in pure cultures by a number of pathologists. Inoculations from pure cultures and the production of typical cankers are reported first by Murrill and later by various others. The writers of this bulletin have separately carried out Koch's four rules of proof many times, in New York and Pennsylvania.

On other species of Castanea

Metcalf (1908 a), after having failed to produce the disease by inoculations on Japanese chestnuts in the field, states that this variety is immune.

Murrill (1908 a), however, found cankers on Japanese chestnuts. Clinton (1913:375), after having failed to produce the disease on a Japanese tree inoculated in sixteen different places, states that this variety shows more or less immunity. Metcalf (1914:16), in his latest publication, says. "The Japanese chestnut is highly resistant, and certain strains apparently immune. . . . . At present we do not know exactly what the Japanese chestnut is; most of the trees that pass under this name in the American market appear to be hybrids with the American or other varieties."

Pantanelli (1911) and Metcalf (1912 b) proved by inoculation that the European chestnuts are not immune.

Murrill (1908 a) found the chinquapin, *Castanea pumila*, attacked.

Morris (Clinton 1913:376) reports that the Chinese and northern Japanese and Corean varieties show decided resistance. Meyer, as stated by Fairchild (1913), finds that the chestnut trees in northern China are not immune, but that they suffer less than do the trees in America. Observations by Morris (1914) and Van Fleet (1914) on the relative immunity of hybrids have been noted above, under the discussion of hosts.

In general, no species nor variety of the genus Castanea has been proved to be immune, but some of the oriental varieties show a certain degree of resistance.

On trees outside the genus Castanea

Fulton (1912:53) reports *Endothia parasitica* on *Quercus alba* and *Q. velutina*, but considers it entirely saprophytic. Clinton (1913:377) adds *Quercus rubra* to the list, but does not believe that the fungus is ever an aggressive parasite on the oaks.

Anderson and Babcock (1913) made cross inoculations, and they discuss this phase in detail. The results of their experiments may be summarized as follows: The fungus was found growing naturally in the woods on *Quercus velutina*, *Q. alba*, *Q. prinus*, *Rhus typhina*, *Acer rubrum*, and *Carya ovata*. Only on the white oak did it seem to have established parasitic relations. (See also Rankin, 1914.) It was isolated from all of these except *Q. prinus*, and when grown in pure culture it appeared identical with the strains from the chestnut. The strains from *Quercus velutina*, *Q. alba*, *Rhus typhina*, and *Acer rubrum* produced typical cankers when inoculated back on chestnuts. The others were undoubtedly *Endothia parasitica*, judging from cultural characters, although no cross inoculations were made. Inoculations with the fungus isolated from chestnut were made on *Quercus prinus*, *Q. velutina*, *Q. alba*, *Q. coccinea*, *Rhus typhina*, *Acer rubrum*, *Liriodendron tulipifera*, and *Carya ovata*. Two trees of Rhus

were girdled and killed by the growth of the fungus. It grew and produced spore horns also, on the wounded tissue at the point of inoculation, on all the others except the maple and the tulip. On *Quercus alba* and *Q. prinus* the cankers continued to spread for several weeks, and fan-shaped mats of mycelium were found under the bark. From the fact that such mats are never found, even on the chestnut, except where the parasite is invading living tissue, it was decided that in these cases, at least, it had established parasitic relations. None of these trees were killed. The fungus was reisolated from all these hosts, and in culture proved to be the same as that isolated from chestnuts.

It may safely be said that at present the canker fungus is not a serious menace to any other forest tree except the chestnut.

Life history

Germination of pycnospores

The pycnospores cannot be made to germinate in pure water. The writers have found the most satisfactory medium for this purpose to be a decoction made by boiling chestnut bark. The spores will germinate also on sterilized twigs of a large number of trees, on various nutrient media, and even on humus alone; but these media are not favorable for observation of the process under the microscope.

The time required for germination varies with the temperature. Fulton (1912:52) states that he found conidia germinated best at a temperature of 60° F., and distinctly less rapidly at temperatures 10° below or above that point. The writers obtained the most rapid germination at 89° F., the shortest time for the appearance of germ tubes being twelve hours. At temperatures ranging from 60° to 75° F., germination occurs in eighteen to thirty-six hours; at lower temperatures the process often requires four or five days. From this it appears that the warm periods of summer are the most favorable for infection by pycnospores. All attempts to produce the disease by inoculating with pycnospores in winter have failed.

The process of germination begins with an enormous swelling of the spores. Spores averaging 1.28 by 3.56 μ before germination, were found, at the end of eighteen hours in chestnut-bark decoction, to average 6.86 by 10.53 μ, the largest observed being 9.05 by 14.48 μ — representing an increase in volume of over one hundred times that of the original spore. A germ tube grows out from one end, and usually this is followed later by a second one from the opposite end. The rate of growth and the manner of septation and branching of the germ tube are best understood by reference to the series of camera lucida drawings of a single spore at short intervals, reproduced in Fig. 90. The swelling of the spores is due, not merely to a mechanical imbibition of water, but also to a process of growth.

Pycnospores stained just before the germ tube is started show that the increase in size is accompanied by active nuclear division, two to six

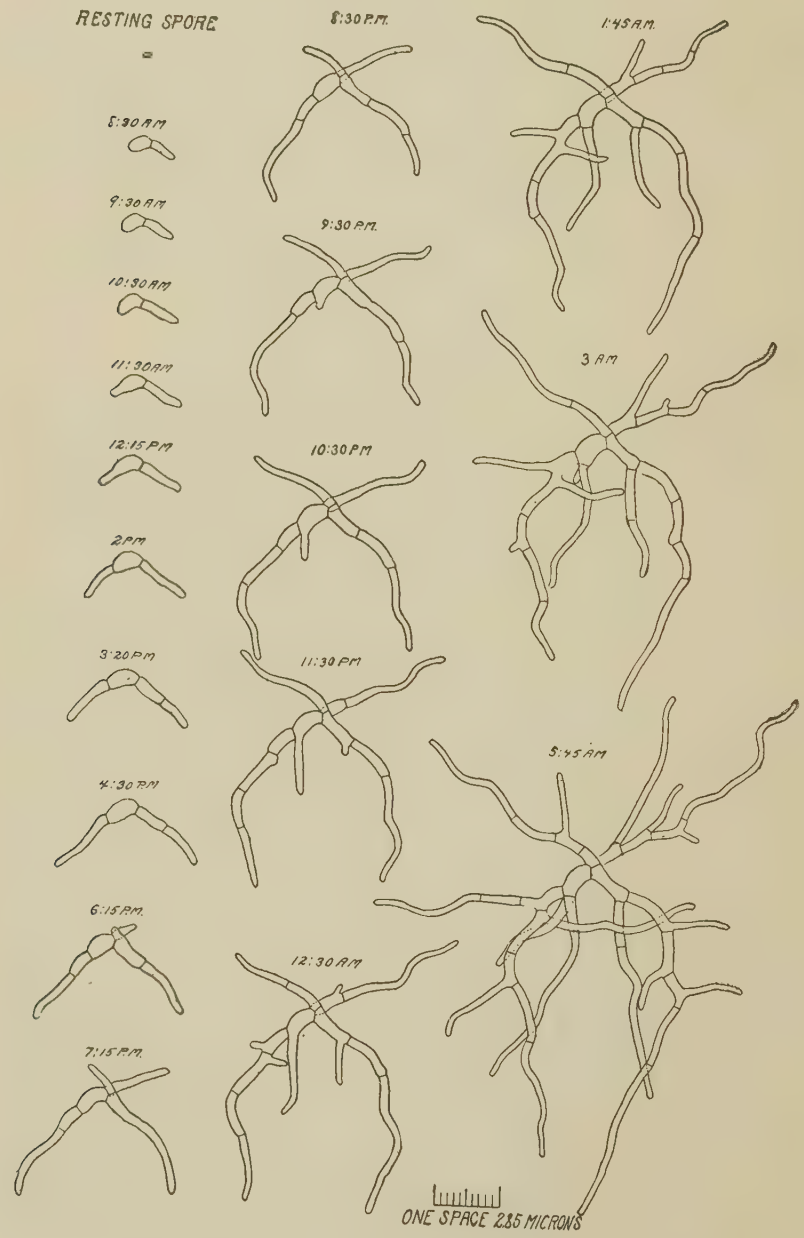

FIG. 90.— *Successive stages in the germination of a pycnospore*

nuclei then being present. The nuclei pass out into the germ tubes almost as soon as they start. The wall, also, has increased in thickness until it almost equals the diameter of the resting spore.

Germination of ascospores

Unlike the pycnospores, the ascospores germinate readily in pure water. They do not require a period of rest, but germinate directly after maturity if placed under proper conditions. The time required for the process to begin after the ascospores are placed in water is much shorter than for pycnospores, being about six to twelve hours at room temperature. In regard to the effect of temperature on germination, Fulton (1912:52) says: "Ascospores germinate best at a temperature of about 70° F., but a good percentage of germination occurs at 85° and 45° F. Even at 38° F. the germination of ascospores was 25 per cent in the first twenty-four hours and reached 70 per cent in three days."

Like the pycnospores, the ascospores swell before germination, but not to so great an extent. The first germ tube usually appears at the end — only occasionally being lateral; the next one comes from the other cell; and these are followed by a second one from each of the cells, making a total of four germ tubes. Their order of appearance, size, and manner of septation and branching are best understood by reference to the drawings of successive stages in Fig. 91. The germ tubes from the ascospores grow much more vigorously than do those from the pycnospores. By sowing ascospores on chestnut-bark agar, mature pycnidia have been produced in five days.

Vitality and longevity of the spores

Pycnospores.— Reasoning from analogy with what is known of the vegetative spores of many other fungi, one would not expect the pycnospores to survive winter conditions; but the fact is quite the contrary. During each month of the winter of 1912–1913, spores were collected in the woods from spore horns, from pycnidia imbedded in the stromata, and from superficial pycnidia on wood, and in every test more than fifty per cent of the spores germinated. It appears, then, that winter conditions have very little effect on the viability of the pycnospores. Heald and Gardner (1913 a) also find that pycnospores can be subjected to freezing temperatures for considerable periods without losing their viability.

The longevity of the conidia varies greatly with the way in which they are kept. Spore horns collected and kept dry in the laboratory and tested each month for germination showed very little diminution in the percentage of viable spores at the end of one year. On the other hand, when the spore horns were dissolved in water and the water was allowed

to evaporate, leaving the spores in a separated condition on the slide, they did not retain their viability longer than a month. The difference

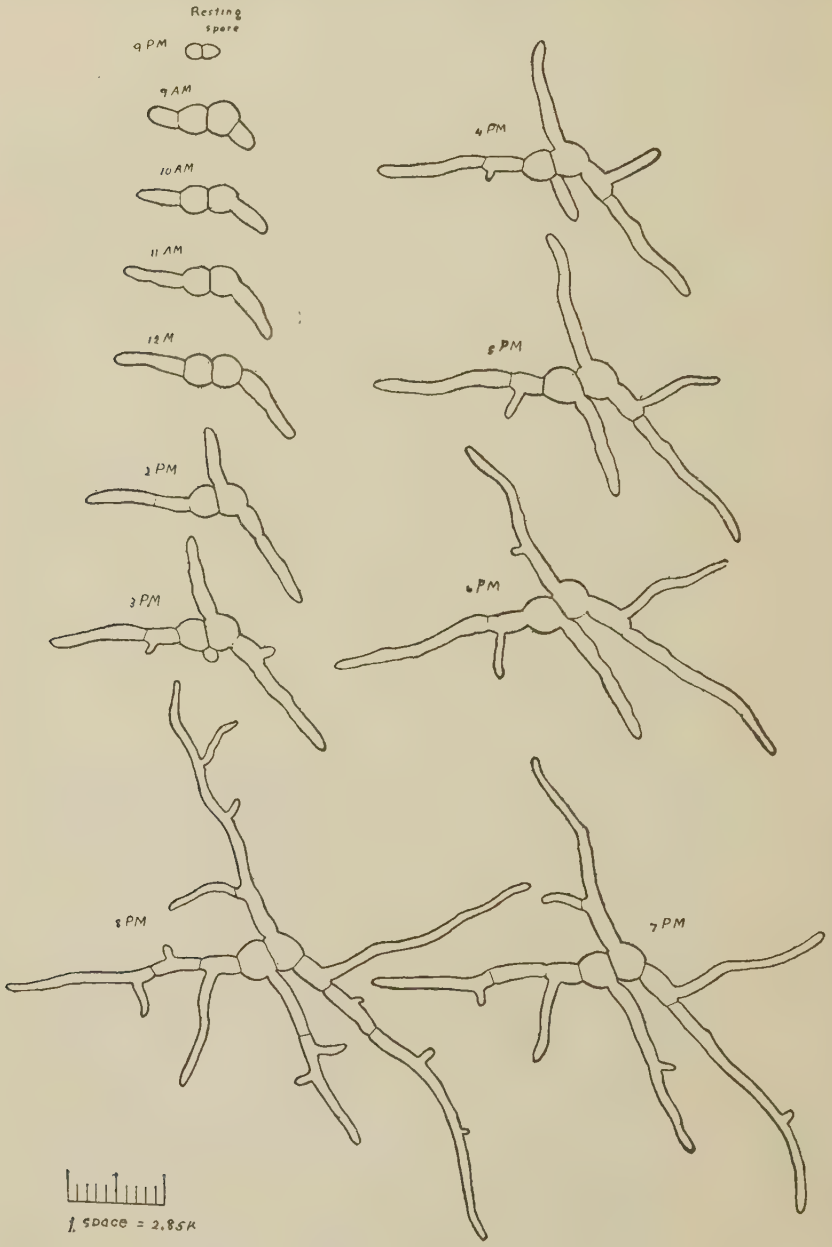

FIG. 91.— *Successive stages in the germination of an ascospore*

has not been explained satisfactorily. It would suggest that possibly the gelatinous substance which binds the spores together in the tendrils may serve also as a protective covering. Gardner (1914), however, finds that when washed down into the soil at the base of the tree the spores retain their viability in every case, in large numbers, until the next rain. Even when such soil is taken into the laboratory and kept dry, he has found the spores still alive at the end of one hundred and nineteen days.

Ascospores.— These were also collected and tested during each month of the winter, but apparently winter conditions had no effect on their viability. Their longevity also seems to vary with the amount of aggregation and exposure. In one series of experiments, ascospores ejected from perithecia were caught on slides and stored in a dry place. Tests showed that the percentage of viable spores decreased each month, until, at the end of five months and six days, none of the spores could be made to germinate. In Bulletin 9 of the Pennsylvania Chestnut Tree Blight Commission, however, it is stated that when the ascospores are washed and then dried they are very sensitive to desiccation, and that " drying alone has been found to kill as many as ninety-four per cent in certain tests." In another series of experiments made by the writers of this bulletin, bark containing stromata with mature perithecia was stored in a dry place and germination tests were made from spores taken from these perithecia each month. At the end of one year very little diminution in the percentage of germination could be seen. In another series of experiments, a large percentage of the ascospores similarly stored germinated after eighteen months, while ascospores from material kept for thirty-one months produced only primary and secondary buds and no germ tubes nor mycelium.

Inoculation and infection

The inoculum.— The disease may be produced on healthy trees by introducing into the bark (1) pieces of diseased bark or wood from other trees, (2) bits of agar or other culture media containing the mycelium in pure culture, (3) pycnospores, either as dry spore horns or suspended in water, (4) ascospores suspended in water or dry. The writers have used all of these in producing hundreds of cankers. The highest percentage of infection was obtained from the first, the next highest from the second; very little difference was noticed in the last two, which have been used more extensively and which best imitate the process as it occurs in nature.

Necessity of a wound.— Murrill (1906 a) failed to secure infection as long as " the thin brown layer of cortex remained intact," and was therefore of the opinion that wounds are necessary; but he also suggested the possibility of lenticels being channels of entrance. Metcalf and Collins (1910) state that " the parasite can enter without visible breaks

in the bark, but wounds form the usual means of entrance." Anderson and Babcock (1913) were unable to produce the disease by inoculating with spores without making wounds.

At Napanoch, New York, glass rings were affixed to healthy, smooth bark, which was then sprayed with a suspension of ascospores. The rings were then closed in order to secure moisture conditions favorable for germination. The purpose of the experiment was to see whether infection could take place through lenticels. Forty-nine inoculations made in this way gave negative results. In another set of experiments the rings were placed over natural cracks — which are abundant on heavy-bark trees in spring and early summer, due to the rapid growth of the trees — and these were inoculated as above. The fourteen inoculations made in this way gave negative results. In a supplementary set of experiments, ascospores were sprayed on one hundred and ninety of these cracks and were not protected in any way. All but four gave negative results. In still another set, twenty pieces of bark containing perithecia were placed so that the ascospores would be ejected during rains into these natural cracks. No infection resulted.

As is indicated later under another head, the fungus appears to be incapable of effecting any change in the cork tissue. It should be kept in mind, also, that even when the bark appears sound it may contain small punctures or abrasions which could easily escape notice; and that, unless the point of inoculation is well protected, wounds may be produced subsequent to inoculation which are not taken into account by the experimenter. It is evident, then, that at most the cases in which the fungus enters without an abrasion must be so rare as to be negligible.

Agents that produce the wounds.— When the disease was first brought to notice, Murrill (1906 a:151–152) suggested a number of agents that might be responsible for the wounds that give entrance to the parasite — such as lumbermen, nut-gatherers, winter injury, the green fly, the twig-borer, squirrels, birds, insects, mice, moles, and rabbits. Later (1906 b) he suggests dead twigs as a channel of entrance, since he often finds these at the center of young cankers.

Metcalf and Collins (1911:10) believe that insects are responsible for most of the wounds: "When the spores have once been carried to a healthy tree, they may develop in any sort of hole in the bark which is reasonably moist. These may be wounds or mechanical injuries, but by far the most common place of infection is a tunnel made by a borer. . . . In many parts of the country where the disease is prevalent there is very direct evidence that bark borers, and particularly the two-lined chestnut borer (*Agrilus bilineatus*), are directly associated in this way with ninety per cent or more of all cases of this disease." Ruggles (1913) points out

that Metcalf and Collins probably had in mind the tunnels produced by the bast-miner, an undetermined species.

Anderson and Babcock (1913) and Rankin (1914) have called attention to the fallacy of basing conclusions as to origin on the wounds found on a canker, since these are quite as likely to have followed as to have preceded the entrance of the parasite. These experimenters made a large number of inoculations in various kinds of wounds, imitating the wounds that are found naturally on the trees. Their results are given in Tables 1 and 2:

TABLE 1. COMPARATIVE VALUE OF DIFFERENT KINDS OF WOUNDS FOR INFECTION. CHARTER OAK, PENNSYLVANIA

Character of wound	Inoculum	Number of inoculations	Percentage successful
Longitudinal slit	Diseased bark	568	93.6
Longitudinal slit	Mycelium from culture	454	89.2
Diagonal slit	Mycelium from culture	25	96.0
Diagonal slit	Dry spore horns	16	93.7
V-shaped cuts	Conidia in water	97	93.8
V-shaped cuts	Ascospores in water	88	94.3
V-shaped cuts	Ascospores shot in dry	1,228	82.5
Artificial borer holes	Mycelium from culture	68	54.4
Natural insect holes	Mycelium from culture	18	0.0
Natural insect holes	Conidia in water	23	0.0
Natural insect holes	Ascospores in water	22	0.0
Peeling down bark	Mycelium from culture	25	88.0
Stab with knife	Ascospores in water	347	36.9
Stab with knife	Conidia in water	81	86.8
Stab with knife	Dry spore horns	96	79.1
Scraping off outer cork layer	Mycelium from culture	25	0.0
Cut stubs	Mycelium from culture	22	81.8
Broken branches	Mycelium from culture	45	71.9
Natural cracks	Mycelium from culture	25	0.0
Gimlet holes	Ascospores in water	135	52.6
Hypodermic needle	Ascospores in water	54	75.9

TABLE 2. RESULTS OF INOCULATIONS MADE IN WOUNDS FROM MAY 1 TO SEPTEMBER 1, 1913, AT NAPANOCH, NEW YORK

Method	Inoculum	Number made	Number infected	Percentage of infection	Average percentage of infection
Slits in bark	Ascospores in water	89	88	99	92
	Conidia in water	116	95	82	
	Mycelium from culture	61	61	100	
	Diseased bark	93	93	100	
	Conidia dry	10	10	100	
	Pycnidia from wood	11	4	36	
Injections	Ascospores	107	74	69	74
	Conidia	56	46	82	
Saw wounds	Diseased bark	50	26	52	52

As to the nature of the wound necessary for infection, they conclude that any kind of wound in the bark deeper that the outer green cortex may furnish an entrance.

The fact that a large proportion of incipient cankers occur about dead twigs, or small branches, has suggested that the pathogen gains entrance to the living tissue through the dead tissue of such branches. But it is usually impossible to tell whether the dying of the twig preceded the entrance of the fungus, or whether the twig died as a result of the canker having been formed about it. Other reasons could be assigned as explaining the above condition. The rough bark around the insertion of small branches remains moist longer than does smooth bark, and thus furnishes more favorable conditions for spore germination. Insect injuries are more numerous at such places and also the bark often becomes cracked above the insertion, thus offering more opportunities of entrance to the parasite.

Age of tree and parts of host affected.— After the first year the age and the size of the tree make no difference in its susceptibility. Not only may cankers be found in the woods just as often on trees an inch in diameter as on those two feet in diameter, but also inoculations on large trees produce the disease just as surely as do those on small trees. Also, there is no difference in susceptibility between the trunks and the branches of any size. Inoculations produce the disease equally well on all.

All inoculations of green leaves have failed. Murrill (1906 a) was unable to produce the disease on green shoots of the first year. Metcalf and Collins also state that green twigs are not affected. Metcalf (1913:365) states: "Late in the season it will readily attack wood of the current year." Anderson and Babcock (1913), however, successfully inoculated first-year green shoots both with ascospores and by inserting diseased bark; Rankin (1914) also produced the disease on first-year shoots by inoculation with ascospores at Napanoch, New York. Yet it is readily apparent that green shoots are less often attacked than are older ones; also the percentage of infection is higher during the latter part of the growing season than during the early summer. All inoculations made by the writers on green burs have failed.

Metcalf and Collins (1910) state that roots are rarely, if ever, attacked, but the pustules of the fungus are commonly found on exposed roots; Anderson and Babcock (1913) were able to grow the fungus on subterranean roots, also, but no typical cankers were produced. It is certain that the roots are not killed by the fungus, because they seem not to lose their power of producing new shoots even when the part of the tree above ground is entirely killed by the parasite.

The orientation of lesions on the trees shows no relation to the points of the compass. Data compiled for nearly a thousand trees taken at

random in New York and Pennsylvania showed no preponderance of cankers on any particular side of the trees. (Anderson and Babcock [1913] and Rankin [1914]).

Effect of season.— Beginning with June, 1912, inoculations were made at Charter Oak, Pennsylvania, in every month of the year. Twenty-five or more inoculations were made each time, with each of the following inocula: (1) ascospores, (2) pycnospores, (3) agar containing mycelium, (4) diseased bark. The spores were inoculated by making a suspension of them in water and inserting a few drops into a stab made with a knife in the bark; the other inocula were inserted into a small slit in the bark. In Table 3 are shown the results for the year, the plus sign meaning that infection resulted and the minus sign meaning that it failed. Where the results are marked positive, over fifty per cent of

TABLE 3. RESULTS OF MONTHLY INOCULATIONS

Inoculum	June	July	August	September	October	November	December	January	February	March	April	May
Ascospores	+	+	+	+	—	—	—	—	—	—	+	+
Pycnospores	+	+	+	+	—	—	—	—	—	—	+	+
Mycelium in culture	+	+	+	+	—	—	—	+	—	—	+	+
Diseased bark	+	+	+	+	+	+	+	+	+	+	+	+

the inoculations produced cankers in every case except in the January inoculations with mycelium, of which only a very small percentage were successful. A similar set of experiments at Napanoch, New York, gave like results. The results indicate that infection from spores does not occur during the six months from October to March, inclusive. Apparently, then, even if spores should gain access to wounds during the winter, infection would not necessarily result. Very little difference between the percentage of cankers produced was noticed during the remaining months of the year.

Development of the canker

If the inoculum is diseased bark or agar containing mycelium, the canker usually begins to show externally in about two weeks after inoculation in summer. If either kind of spores are used, the canker does not show until after three to five weeks. A longer time is required in the cool months of spring and autumn. The canker appears first as a darker area about the point of inoculation. In dry periods this affected part soon sinks and at the same time the bark takes on a reddish color. In

this case the limits of the canker are easily distinguished on the surface; in rainy seasons the outline at first is not sharp, but if the canker is cut into with a knife a sharp line of demarcation will be found between the brown, diseased bark and the white, healthy, inner bark.

When the spores have germinated in a wound, the germ tube thrives on the injured and dead cells until it has produced a mass of mycelium. Then, gradually accumulating strength as it increases, the mycelium *en masse* pushes out into the living tissue of the bark. Single threads do not seem to possess the power of penetrating alone among the living cells, but the invasion is accomplished by the force of mass action. Starting from a narrow point, the hyphæ grow out in ray-like bundles, completely destroying parenchyma and collenchyma and cambium cells as they go. The rôle of toxins or enzymes in this process has not been investigated. All the rays starting from a single point are contiguous, forming the fan-like mats previously mentioned.

Rate of growth of mycelium.— The rate of growth of mycelium under natural conditions in the tree can be roughly measured by the increase in size of the cankers. The growth varies with the temperature, being the most rapid during the summer months; but the mycelium does not always remain dormant during the winter, as will be shown. For twelve consecutive months at Charter Oak, Pennsylvania, cankers were outlined with a painted white line at the end of each month and the average rate of growth was computed. A second experiment of a similar nature was conducted at Napanoch, New York. The data on these two experiments are given in Tables 4 and 5. These tables give only the rate of growth

TABLE 4. MONTHLY RATE OF GROWTH IN DIAMETER OF CANKERS AT CHARTER OAK, PENNSYLVANIA

Month	Number of cankers	Average growth per month (in centimeters)
1912		
June	31	1.88
July	200	2.78
August	186	2.83
September	140	1.85
October	53	1.92
November	27	0.00
December	27	——
1913		
January	89	0.51
February	89	0.00
March	84	0.7
April	21	1.1
May	41	2.4

TABLE 5. AVERAGE RATE OF GROWTH OF CANKERS DURING SUMMER MONTHS, AT NAPANOCH, NEW YORK

Date when inoculations were made	Number of inoculations	Growth by periods of four weeks (in centimeters)				
		May	June	July	August	September
May 2	60	1.35	1.75	3.0	2.4	2.3
June 1	48	.....	2.5	1.72	3.0	.....
June 11	16	.....	.....	2.0	1.9	.....
July 3	41	.....	.....	1.75	.....	.....
July 11	105	.....	.....	2.0	1.7	1.7
August 1	11	.....	.....	.....	2.1	.....
Average	.....	1.35	2.08	2.1	2.2	1.9

of the canker around the tree, the increase in length being considered not so important. It appears that the mycelium continues to grow even during mild periods of the winter, such as were frequent during January of 1913.

As a check on the above data, agar plate cultures were kept out during the winter and growth was recorded during each warm period. In this connection, Shear and Stevens (1913 a:9) note that the minimum temperature for growth of the fungus in culture is 9° C. (48° F.), which temperature was exceeded on ten different days during January.

Even the most rapid growth, as recorded above for the summer months, is less than one millimeter a day. But on artificial media a growth of three millimeters a day is not unusual. Mycelium also spreads at a much more rapid rate in the dying bark after a tree is cut.

Vitality of mycelium.— The mycelium does not seem to be injured in the least by freezing. It remains alive in all parts of the canker during the winter. Cultures were kept frozen for a month at a time, and resumed growth naturally on being brought back into the laboratory. The mycelium is not readily killed by desiccation. Bark removed from a canker and kept perfectly dry in the laboratory for ten months yielded quite as successful isolations as did fresh bark. Chips of diseased bark left on the ground in the woods were found to contain living mycelium one year later. This remarkable vitality of the mycelium is one of the factors that make the disease difficult to control.

Development of pycnidium.— The pycnidial stage usually appears on the cankers in three to six weeks after inoculation. The very earliest stages in the development of the pycnidium are not readily found and studied in the bark, but when the fungus is grown in Van Tieghem cells in drops of agar the process can be easily observed under the microscope. At room temperature it begins in less than a week, in the following manner:

At a certain point on a hypha, short cells are formed by the laying down of new septa. These cells increase in diameter and in amount of contents and each one sends out short septate branches (Fig. 92, a and b),

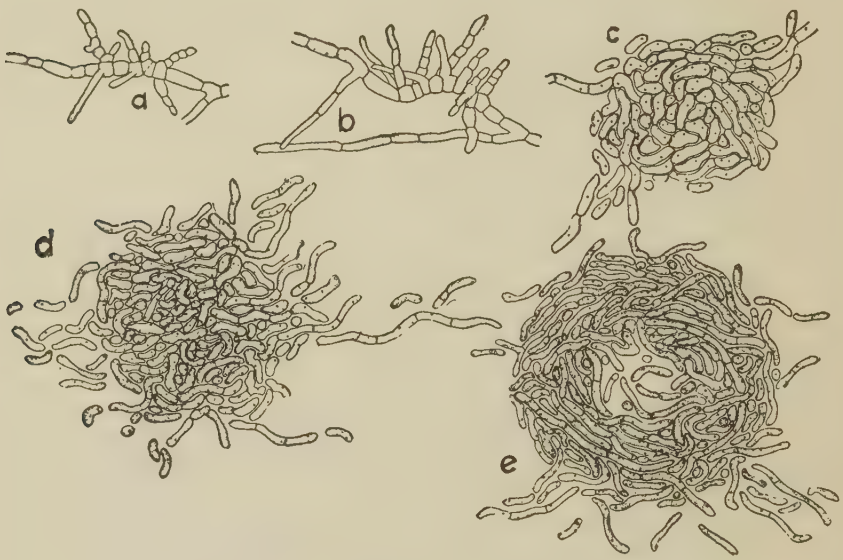

FIG. 92.— *Development of pycnidium*

a and b, Very earliest stages observed in Van Tieghem cell cultures
c and d, Cross sections of pycnidium on agar before the beginning of the cavity
e, Cross section when the cavity first becomes apparent

the individual cells of which in turn put out other short branches, until the whole structure has the appearance of a witches' broom. Other hyphæ, and more distant branches of the same hypha, grow toward it and mingle with its branches. This tangle of hyphæ soon becomes so dense that a surface view does not show what is occurring on the inside, and later stages have to be studied from serial sections after imbedding, sectioning, and staining. Cross sections at this time and for some days later show that the mass is merely an increasing solid ball of hyphæ, which are all alike and densely interwoven but not anastomosed in any way (Fig. 92, c and d). A little later the hyphæ at the center appear

looser (Fig. 92, e) and those branches that extend into this loose central area begin to cut off regular cells (pycnospores) from their apices. More branches (conidiophores) now grow in and cut off spores from their apices, until the wall, which constantly recedes as the pycnidium becomes larger, is completely lined with the brush-like hymenium (Fig. 86, page 559) and the cavity is packed with spores. Meanwhile an ostiole is formed by the loosening of the hyphæ at the apex, and the spores are forced out through it in a gelatinous mass. The cells of the loose wall seem never to divide meristematically, and the wall in this type never appears pseudoparenchymatous.

The first outward indication of the formation of pycnidia on the young canker is the appearance of numerous small, raised, smooth blisters just back of the advancing edge of the lesion (Plate XXXVIII, Fig. 1). They show no relation to the lenticels. Under each blister is the beginning of a single pycnidium. At this stage the pycnidia are more or less globose cushions with a moist, gelatinous appearance; about half of the pycnidium is imbedded in the disintegrating collenchyma tissue, the other half projects upward and raises the cork layer to form the pimple. There is no stroma at this time, but each pycnidium is early surrounded with a fringe of loose mycelium which is the forerunner of a stroma. A cross section of this hygrophanous cushion shows it to be a closely wound ball of hyphæ corresponding to the stage represented in Fig. 92, c and d. It increases in size and develops into a mature pycnidium just as the process on agar was described above, pushing up the cork layer and finally causing it to rupture at the tip, from which point the spores are forced out. Meanwhile the stroma is forming about the pycnidium. So far as observed, the stroma never precedes, but always follows, the early stages in the development of the pycnidium. With the active increase in the amount of stromatic tissue which is constantly added from below, the pycnidia are pushed out in the top of the stroma through the cork layer. Meanwhile they continue to increase in size and become irregular in shape. (Fig. 84, page 558, and Plate XL, Fig. 1). Often the entire stroma is found honeycombed with numerous communicating lobulated chambers.

Time of production of pycnospores.— In Pennsylvania during the seasons of 1912 and 1913 the spore horns first began to appear on the cankers about the middle of April; after that they could be found at any time during the summer, being especially abundant after periods of rain. During the season of 1912, on cankers produced by inoculation in the spring, the tendrils were abundant after each rain period until the latter part of the summer, when the perithecial stage began to develop; after that, few spore horns were found on the cankers. Heald and Gardner (1913 a) state that pycnospores are produced in enormous numbers during

the winter months. Fresh spore horns are not seen during the winter, but this may be due to the fact that they are produced at such a slow rate that they are washed away before their size makes them noticeable.

Development of perithecium.— The beginning of the perithecial stage is accompanied by a marked increase in the size of the stroma, which now pushes off more of the cork layer and not only fills up the enlarged rent but also grows out over the torn edges so that they are included in it (Fig. 85, page 558). The stroma takes on an erumpent, superficial appearance (Plate XXXVII). This change has been observed within eight weeks after inoculation; on trees inoculated in June the stromata have been found in August.

FIG. 93.— *Stages in the development of the carpogonium. The larger, heavily shaded cells at the center are the ascogonial cells, with the cells of the enveloping hyphæ about them. The last figure shows the continuation of the ascogonium into the trichogyne. All figures are drawn to the same scale*

The primordia — beginnings of the perithecia — arise in the base of the stroma or even among the outermost cells of the host. Each primordium consists of two to five large, prominent cells arranged in a circle or a spiral (Fig. 93), closely invested by a sheath of large hyphæ. The central prominent cells form the ascogonium, and the investing hyphæ will here be called the envelope. The ascogonium, which is an enlarged single hypha, is continued up to the surface of the stroma as a prominent thread, the trichogyne. There may be as many as a hundred primordia in a single stroma, but only about one fourth of them ever reach maturity; the others degenerate at various stages of development.

The ascogonial cells are elongated-oval, slightly curved so as to fit the segment of the spiral, deeply constricted at the septa, and only loosely

connected. They are filled with dense protoplasm and contain more numerous and more prominent nuclei than do the cells of the envelope. The cells of the trichogyne resemble those of the ascogonium in every way except that they are narrower and are not curved nor so loosely connected. The trichogyne is apparently a useless organ in the formation of the perithecium. During the development of the latter it degenerates and disappears.

The highest stage in the development of the ascogonium is shown in Fig. 93. From this time on it begins to degenerate. The dense protoplasmic content gradually becomes thinner, and later there are only ragged bridles of protoplasm across the lumen and irregular masses around the walls (Fig. 94) or else the entire contents draw up into a misshapen mass. But the behavior of the enveloping cells is quite the contrary. Their contents now become more dense, and their nuclei more prominent and apparently more numerous. Up to this time the individual hyphæ can be traced and open spaces are apparent between them; but now they have increased both in size and in number and have filled up the intervening spaces. They appear as a pseudoparenchymatous tissue instead of as a coil of hyphæ.

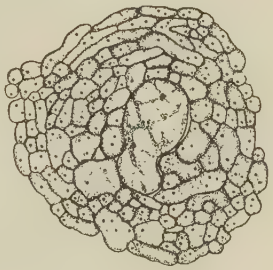

FIG. 94.— *Cross section of the developing perithecium just as the ascogonial cells (one large one shown at the center) are beginning to degenerate and the enveloping hyphæ are taking on a pseudoparenchymatous appearance*

Before degeneration the ascogonia probably give rise to ascogenous hyphæ, but these are very quickly cut off by septa and are indistinguishable among the enveloping hyphæ. The ascogonial cells are soon crowded out of shape by the growth of the enveloping cells, and in later stages they appear only as misshapen masses wedged between the other cells. The whole primordium now increases rapidly in size and takes on the appearance shown in Fig. 95. The beginning of the neck is shown at the top of the figure. Next, the wall is differentiated from the large-celled core. A cavity is formed by the breaking-down of some of the core cells, but is soon almost filled by paraphyses which arise from the bottom of the cavity (Fig. 88, a, page 561). The young asci grow up between the paraphyses, arising from a system of hyphæ which are presumably the continuation of the branches of the ascogonial cells. Eight bicellular spores are formed in each of the clavate asci. The hyphæ that initiated the formation of the neck now push toward the surface, leaving a canal through the center (Fig. 88, b). Branches of these hyphæ extend into the canal to form the periphyses (Fig. 88, c). Meanwhile the cavity

has become completely filled with maturing asci and the perithecium is now ready to discharge its spores.

Time of development of perithecia.— On cankers produced by inoculation during spring and summer, mature perithecia are developed in early autumn. It is evident, however, that this is not the only season at which they may be produced, because there is no time of the year at which they cannot be found in abundance, ready to eject spores if the proper conditions of moisture and temperature are supplied.

FIG. 95.— *Cross section of the young perithecium before the differentiation of the wall and the core cells, showing at the center the shriveled remains of an ascogonial cell and at the top the beginning of the neck*

Ejection of ascospores

One of the writers of this bulletin (Rankin, 1912 a) discovered that during rain periods the ascospores are ejected with some force into the air from the ostioles of the perithecia. The asci, which usually fill the perithecium, are pushed up through the neck when abundant free water is added to the stroma. This expulsion of the asci is largely due to the swelling pressure of the asci. A dry ascus with its spores (Fig. 89, b, page 562) occupies only about one half the space occupied by an ascus after water is added (Fig. 89, c and d).

Prepared sections of perithecia fixed during the process of ejection of spores show that the spores remain in the asci almost to the tip of the neck. Since the asci themselves are never ejected into the air, it follows that they must burst and liberate the spores when they arrive at the surface of the film of water. The ejection may be observed with a hand lens. The only visible phenomenon is the sudden and regular breaking of the film over the ostiole. This gives for the moment the impression of a point of light. The entire contents of the ascus are ejected at once into the air.

Rankin (1914) has drawn the following conclusions as to the process of the actual ejection of the spores from the asci: The imbibition of water results in the asci being forced out through the ostiole, where,

if a film of water is present, the walls begin to gelatinize. Undoubtedly, as more and more asci are pushed out, some arise in the water to the surface and may even project above the surface, due to the disturbance. It might be expected, then, that the increased pressure on the ascus wall due to the swelling of the contents, at a time when the wall is gelatinizing, will result in the sudden rupture of the wall when the ascus arises to or above the surface of the water. Since the lower part of the ascus wall is the thinner and gelatinizes first, freeing the spores, and since no change suggesting pore or other type of apical dehiscence has been found, it is presumed that what proves to be a successful method of ejection is due merely to a combination of physical factors, not to any structural arrangements.

Saprophytic growth

Endothia parasitica may live indefinitely as a saprophyte. In this condition it is not so restricted in its feeding habits as is the case otherwise. Where it is found growing on trees outside the genus Castanea, it usually exists there only as a saprophyte. It may be grown in pure culture on sterilized twigs of almost any of the common forest trees. It has also been grown on a wide range of culture media. Anderson and Babcock (1913) report cases in which trees with scattered cankers were cut and were left lying on the ground during the summer; in the autumn the fungus was found to cover the entire trunk. Also, they found that the fungus spreads rapidly in fire-killed bark. When it grows in dead bark, no canker-like area is formed, and also the mycelium does not progress by the production of fan-like mats but by single threads. The fungus spreads through dead bark many times more rapidly than through living tissue. Anderson and Babcock made successful inoculations on dead bark with ascospores and with mycelium, and on dead leaves and dead burs with ascospores. The fungus grew and produced pycnidia on all of these. Collins (1913) also found the fungus growing saprophytically on burs. Apparently its growth is checked only by failure of the moisture supply.

In order to determine how long the mycelium lives on logs after they have been cut, both with the bark on and with it removed, and also on the stumps from which the logs are cut, and whether the fruiting stages are produced under these conditions, the following experiment was performed: Thirty-two trees, some entirely dead and some still living, were felled, cut into seven-foot logs, and allowed to remain lying on the ground. Some were peeled, others were left with the bark attached. Also some of the stumps were peeled. Isolations were made from the interior of the bark and the wood — care being taken to avoid getting any spores of the fungus in the cultures — during each month for one

year. In Table 6 are shown the results of the last set of isolations, at the end of one year; the table shows how well the fungus may maintain itself as a saprophyte. Pycnidia were formed in countless numbers on the peeled logs and on the cut ends of the stumps and the logs. Stromata and perithecia developed on some of the unpeeled logs, but none developed on the peeled logs. The writers have on several occasions isolated the fungus from woodpiles, rustic woodwork, and the like, where the trees were said to have been cut several years previously.

TABLE 6. RESULTS OF ISOLATIONS FROM LOGS AND STUMPS ONE YEAR AFTER TREES WERE CUT

Trees dead when cut	Number containing live mycelium
15 logs, peeled	11 logs
58 logs, not peeled	46 logs
4 stumps, peeled	4 stumps
13 stumps, not peeled	11 stumps
Trees alive when cut	
3 logs, peeled	1 log
38 logs, not peeled	32 logs
1 stump, peeled	1 stump
9 stumps, not peeled	8 stumps

Where the moisture conditions are favorable, the fungus is often found growing luxuriantly in the bottom of piles of chips where diseased trees have been cut. It has also been proved, by inoculation with conidia, that the fungus will grow and produce pycnidia even on humus such as is found about the base of the trees.

Dissemination

That this fungus spreads with astonishing rapidity is a matter of common observation. Not only does it spread quickly from one tree to those standing close about it, but it is remarkable for its long jumps across country. It suddenly appears in a neighborhood far ahead of the main line of advance, without there being another diseased tree for a distance of many miles. Hundreds of these isolated spot infections have come to notice and have given rise to much speculation as to their origin. After the disease has once appeared in a locality it is usually a matter of only a few years until the neighborhood for miles around is thoroughly infested. What agents are responsible for carrying the causal organism from one tree to another, and thus causing this rapid spread? Many answers have been suggested, but unfortunately most of them have been mere conjectures not based on experimental data.

Most of the early writers confined their attention to the pycnospores, and it has only been within the last few years that attention has been called to the fact that the ascospores also are instrumental in the spread of the fungus. Murrill (1906 a) suggests the agency of wind, squirrels, birds, insects, mice, moles, and rabbits, and later (1906 b) rain—the last only carrying the spores from one part of a tree to other parts. Hodson (1908:5) says, in regard to the dissemination of the pycnospores:

"Wind is probably the principal agency, but the spores are no doubt carried by animals, birds, insects and by shipment of infected material.

"The disease spreads locally through the gradual distribution of the spores from tree to tree, and at a distance chiefly through the shipment of infected material, such as nursery stock, bark, nuts, and other products. There is a possibility that long-distance infection is also effected by means of migratory birds."

Metcalf and Collins (1911:9) state: "As both kinds of spores appear to be sticky, there is no evidence that they are transmitted by wind except where they may be washed down into the dust and so blown about with the dust. The spores are spread easily through short distances by rain; particularly they are washed down from twig infections to the lower parts of the tree. There is strong evidence that the spores are spread extensively by birds, especially woodpeckers, and there is also excellent evidence that they are spread by insects and by various rodents, such as squirrels. The disease is carried bodily for considerable distances in tan bark and unbarked timber derived from diseased trees. One of the most prolific sources of general infection has been the transportation of diseased chestnut nursery stock from infected to uninfected localities."

One of the writers of this bulletin (Rankin, 1912, a and b) called attention to the fact that the ascospores are forcibly ejected into the air, and that these can be caught up by the wind and carried for considerable distances and may well be responsible for a large part of the infection. Fulton (1912:51) reports experiments in which he tried to dislodge spore horns by a strong blast from an electric fan. He found that bits of the spore horns were sometimes broken off and carried for short distances, but were too heavy to be carried for great distances.

Anderson and Babcock (1913) have considered the problem of dissemination more in detail than have the other writers. Unless otherwise mentioned, the matter considered below is taken from their bulletin.

Man

Murrill (1908 a) was the first to call attention to the danger of spreading the disease into new localities on diseased nursery stock. Metcalf and Collins (1909:49) gave especial attention to this phase of the matter,

and believe that it is largely responsible for the long-distance jumps of the disease. They state:

"It becomes more and more evident as this disease is studied that diseased nursery stock is the most important factor in its spread to distant points. In that part of the country where it is already well established in the native chestnuts its progress is rapid and sure, but there is no evidence at present that it is able to pass to remote districts, tens or hundreds of miles away, except on diseased nursery stock. Of course it is conceivable that the spores are carried by birds. Such distribution would, however, follow in general the great lines of bird migration north and south and hence would not be an important factor in the western spread, except locally. During the summer of 1908 nearly every chestnut nursery and orchard of importance in the Atlantic States north of North Carolina was visited, and very few were found free from the bark disease. Several cases were observed where the disease had obviously spread from the nursery to adjacent wild trees."

Five prominent cases of spot infections far ahead of the main line of advance in western Pennsylvania were traced to nursery trees or scions shipped there from infested territory.

In order to see whether the fungus could be carried on tools that were first used on diseased trees and then on healthy ones, thirteen cuts were made on healthy trees with an axe after having chopped into diseased logs with this ax. Cankers developed later about twelve of these. One of the writers performed an experiment of a similar nature, in which a saw was first used in sawing diseased wood and then in sawing off healthy branches. Cankers were developed about twenty-seven of the fifty stubs treated in this way. There is no doubt, then, that the disease may be spread by pruning saws, axes, climbers, and other tools used about the trees.

Metcalf (1913) and others have also suggested the possibility of the fungus being carried for long distances by the shipping of ties, poles, tanbark, and the like. The experiments previously described in treating of saprophytic growth show the remarkable ability of the fungus to maintain and propagate itself under saprophytic conditions. The writers of this bulletin also found that perithecia retain their ability to eject spores for at least seven months under perfectly dry conditions. There is no reason why the disease cannot start in a new locality to which affected logs are shipped, if in that locality the logs are placed near growing chestnut trees. It must be admitted, however, that no case of outbreak of the disease in a new locality has ever been traced to the shipment of such material.

Insects

Insects are found in great numbers both in and on chestnut bark, and the possibility that they may crawl over sticky spore horns and carry spores away to deposit them in wounds, and thus start new cankers, has occurred to the majority of writers who have mentioned dissemination. It is reasonable to believe that many infections do occur in this way, but the mere statement of the probability without experimental data is not convincing; and unfortunately such data are meager in the literature.

In order to prove that an insect — or anything else, for that matter — is a direct agent of dissemination, three points must be demonstrated: (1) that the insect is in the habit of visiting cankers and actually carrying away spores on its body; (2) that it deposits these spores in wounds favorable for germination; (3) that infections do result from wounds inoculated in this way. To induce insects to crawl over damp spore horns or active perithecia and then demonstrate the presence of spores on their bodies, is no proof that they are in nature responsible for the spread of the disease. Ruggles (Penn. Chestnut Tree Blight Com., 1914) has shown the fallacy of this argument. After allowing ants and about twenty other species of insects to run over spore horns and active perithecia, he was able to isolate spores of Endothia from all of them. Yet he proved to his own satisfaction that ants are not responsible for infection. The following is from the summary of his work in Bulletin 9 of the Pennsylvania Chestnut Tree Blight Commission: "Two rooms were set off in an insectary. The inner of these two rooms being thoroughly sterilized was called the sterile room, the outer room was called the blighted room. In the latter as much blight material as could be obtained of the kind required was kept and placed on the ant table, where three colonies of ants made their homes. From the table in this room the ants were allowed to run through a glass tube to sterile seedlings in the sterile room. . . . The result of the experiment was that with the exception of a few dried leaves on each tree which were chewed or worked on by the ants, the trees in the sterile room are as healthy as when first placed on the table to be run over by the ants. The indication, therefore, is that ants are not responsible for blight infection."

Studhalter (1914) also reports the presence of spores on the bodies of twenty-four out of seventy-five insects after they had run over cankers or had been taken directly from diseased trees in the field.

Indirectly, insects may be connected with the spread of the disease by making wounds in the bark where spores may gain entrance after having been carried by some other agent. Anderson and Babcock (1913) and Ruggles (Penn. Chestnut Tree Blight Com., 1914) are of the opinion that this is the most serious way in which insects are related to blight

dissemination. Ruggles states that in some places in eastern Pennsylvania, 86 to 93.8 per cent of all infections were in wounds produced by the seventeen-year cicada. The bast-miner larvæ, which are present in great numbers on smooth-bark trees, have often been said to be responsible in this way. Ruggles states that fifty per cent of the infections in smooth-bark trees originated in the exit holes of these insects. He also suggests their responsibility for the numerous crotch infections, since they oviposit near crotches. Anderson and Babcock, on the other hand, were unable to produce infection by making inoculations in exit holes of these insects.

The pustules of the fungus are often eaten by some insects, the most common of these being *Leptostylus maculata;* but experiments have proved that the spores, after being eaten, are digested and do not serve to spread the disease. Craighead (1912) reports five different species of insects which he found eating the pustules, but believes that they contribute to the control of the disease rather than to its spread. The United States Bureau of Entomology has made much of these discoveries and has suggested that these beneficial insects may be a large factor in the final control of the disease (Pennsylvania Chestnut Tree Blight Commission, 1914). Ruggles, however, shows that the insect mentioned above, *Leptostylus maculata*, carries enormous numbers of spores on its body, and suggests that it is more injurious as a disseminator of spores than beneficial as a destroyer.

Metcalf and Collins (1911) state that in many parts of the country the two-lined chestnut-borer (*Agrilus bilineatus*) is directly associated with ninety per cent of all cases of the disease. It was to the so-called bast-miner, however, that they referred, rather than to the borer mentioned (Ruggles, 1913).

Davis (Penn. Chestnut Tree Blight Com., 1913:48) attributes to ants seventy-five to ninety per cent of the cases of infection in the locality where he was working. The grounds for the statement are the fact that no summer nor winter spores could be found and yet the disease continued to spread, and also the fact that ants were found carrying mycelial threads of the fungus. The anomalous condition of trees in an affected area not producing any spores, and Davis' proof that the mycelium carried by the ants was really that of *Endothia parasitica* and that it was deposited in wounds favorable for infection, require further explanation.

Rain

Rain dissolves the mucilaginous matrix of the spore horns, and the pycnospores are washed down the trunks where they lodge in wounds and produce cankers. They may also be splashed to other trees that are in close proximity to diseased ones. This accounts for the fact that

most trees with cankers above are also diseased about the base. Also, ascospores, which sometimes ooze out instead of being forcibly ejected, may be carried down in this manner. It was proved by a large number of inoculation experiments that either ascospores or pycnospores suspended in water in this manner and introduced into wounds will produce a high percentage of cankers (Tables 1 and 2, page 571).

In another set of experiments, wounds were made beneath cankers on which there were only spore horns. The cankers were sprayed with an atomizer and the water containing the spores was allowed to run down into the wounds. Of twenty-three wounds treated in this way, sixteen developed cankers later. There is no doubt, then, that rain is an important agent in spreading the spores to different parts of the same tree. Rain also plays an important part in bringing about the conditions necessary for the ejection of ascospores.

Birds

It seems reasonable to believe that birds alighting on cankers and picking at them carry spores to other trees and deposit them in wounds where they may germinate and produce cankers. Anderson and Babcock (1913) shot birds during the summer, but no spores could be isolated from them. Heald and Studhalter (1913) tested the washings from thirty-six birds of nine different species, all of which were shot on diseased parts of trees between February and May. Downy woodpeckers, nuthatches, golden-crowned kinglets, sapsuckers, brown creepers, and juncos were found to carry enormous numbers of pycnospores, the smallest number per bird being 5655 and the largest 757,074. No ascospores were found on the birds.

There can be little doubt that birds aid in the dissemination of the fungus.

Wind

Unlike the agents mentioned heretofore, the wind as a disseminator has to do mostly with ascospores. In a number of the earlier contributions to the literature it is stated that the summer spores are blown about by the wind; but this belief was gradually abandoned when it was pointed out that the spore horns are so hard when dry that they cannot be broken even by a strong blast, and when wet they are of a sticky nature and can hardly be detached by the wind. Metcalf (1913:366), however, still believes that under certain conditions they may be carried by the wind, for he says:

"It is conceivable that they may be blown by the wind as far as rain or spray is blown or, mingling with the dust at the foot of the tree, be blown about with the dust."

Experimental data are not cited.

The writers performed experiments in order to determine the conditions necessary for ejection, rate of ejection, spore content of the air, ability of wind-blown spores to produce infection, and the like.

Ascospores are not peculiarly winter spores, but they may be found maturing and ready for ejection at any time of the year. Experiments by Heald and Gardner (1913 a), however, indicate that they are not ejected during the winter. These experimenters found that after the stromata were subjected to low temperatures they did not resume ejection of ascospores again for three or four days after being brought into favorable temperatures, and that the minimum temperature for spore ejection varies from 52° to 60° F. Experiments show that during the summer spores are ejected only during and shortly after rains — in fact, just as long as the bark remains wet. This may be a half hour or it may be more than a day. The same stroma may resume expulsion any number of times during the summer. Alternating periods of abundance of moisture and desiccation were not found to interfere with the ability to eject spores. In case the bark remains wet continuously, it was found that a single stroma would continue uninterrupted ejection for twenty-six days. One case is reported in Bulletin 9 of the Pennsylvania Chestnut Tree Blight Commission, in which bark containing ascospore pustules continued to expel ascospores for over six months (in the laboratory). The rate of ejection from a single ostiole was determined to be one ejection for each two seconds, that is, about four spores a second. This would be 14,400 spores an hour from a single perithecium. The maximum height of ejection found was twenty-two millimeters. Horizontally, from a stroma two centimeters above an agar plate, spores were ejected to a distance of eighty-nine millimeters on the plate, the experiment being performed under a bell jar.

The spore content of the air was determined by two methods. The first was the usual aspirator method used in quantitative analysis for bacteria. By this method no spores could be found in the air during dry weather, but when the air within a few feet of wet cankers was tested there was an average of four and three tenths spores per liter. The second method was the exposure of sterile agar plates at varying distances from moist cankers. When these plates were exposed for a short time close under a canker, as many as 9000 spores were caught on one plate, but the number decreased as the distance between the canker and the plates was increased. Spores were easily secured at a distance of fifty-one feet with a moderate wind blowing from the diseased bark toward the plates on a level with it. No greater distances were tried; but if spores can be so easily caught at that distance on a ten-centimeter plate in a moderate wind on a level

place, one can well imagine their being blown for miles in a strong wind from tall trees and from the tops of the mountain ridges where the disease is frequently found. Heald, Gardner, and Studhalter (1914) were able to catch the spores over three hundred and eighty feet from the nearest tree.

Having proved that the spores are ejected all through the summer and that they are carried about in great numbers by the wind, only one link of a solid chain of evidence was missing: that was to prove that such wind-blown spores produce cankers when they fall into wounds. In the first series of experiments in order to determine this point, bark containing active perithecia was supported within a few inches of sterile wounds in healthy trees for a short time and then the wounds were wrapped with cotton in order to exclude later inoculation. Of the 1395 wounds treated in this way, seventy-eight per cent developed cankers. In the next set of experiments, the distance was increased to one to four feet and a wind was artificially produced by bellows. Seventy-four per cent of the 185 wounds treated in this way had cankers about them later. Wounds treated in the same way but not exposed to shooting perithecia remained uninfected.

In another experiment, groups of trees were selected in which some had cankers on them while others were free from disease. Sterile wounds were made in the healthy trees and protected by fine-meshed wire screen and cotton bands, so that it was believed that spores could not be introduced in any way except by the wind. The cankers on the diseased trees were frequently drenched in order to induce spore ejection. Of the 559 wounds treated in this way, 114 developed cankers.

The results of the above experiments lead the writers to believe that wind is an important factor in the spread of the disease.

Other animate agents

It is easy to believe that the sticky pycnospores remain attached to the feet of animals, such as squirrels, mice, and numerous others which run over the cankers. But after the spores are isolated from them, as undoubtedly they could be, it would still be necessary to prove that such spores are deposited in wounds where they produce infection. Statements made with respect to the agency of animals in dissemination should be supported by more complete chains of experimental data than have been commonly adduced.

It can be said at present only that it is possible that the fungus is spread by such means.

PATHOLOGICAL HISTOLOGY

Keefer (1914) reports detailed microchemic and histologic studies of the effect of the fungus in the bark and the wood of the tree. He

finds that the outer and internal cork layers, sclerenchyma (bast fibers and stone cells), and crystal-containing cells are not in any way affected. All tissues composed of cells having more or less pure cellulose cell walls are destroyed (except medullary rays). The collenchyma and the thin-walled parenchyma of the primary cortex, the parenchyma of the pericycle, the sieve tubes and the phloem parenchyma of the bast zone, and the fascicular cambium, are enumerated as the tissues that are in most cases wholly destroyed and replaced by the fans of mycelium. It was found that cellulose cell walls became partially lignified in proximity to the advancing edge of the mycelium fan. The degree of lignification, however, is not sufficient to furnish immunity of the tissues to the action of the fungus, as seems to be the case with the more completely lignified tissues, such as the sclerenchyma. The cells of the medullary rays in the bast zone are partial y lignified, but the cells are not individually affected or broken. There is an increase in the number of medullary ray cells, due probably to a stimulating action by the fungus. The original cells divide and redivide until great masses of new cells are formed, so that there is a crowding of the phloem tissues and a radial and tangential separating of the segments of the bast zones. This is the histologic change, evidently, which accounts for the slight, and at times pronounced, hypertrophy of the cankered area. The destruction of the various tissues of the bark is explained on the basis that the cells are broken and destroyed by the mechanical action of the advancing mats of mycelium. Since these are the only tissues affected to any extent, the fungus must utilize some of the material for food. Whether or not enzymic activity precedes or follows the apparent mechanical destruction of the tissue was not determined. Until the enzymes and the toxins of the fungus are studied, the actual biologic relation of the host and the parasite cannot be accurately determined.

CULTURAL STUDIES

Media

Endothia parasitica grows luxuriantly on a wide range of culture media. The writers, as well as other investigators, have grown it on sterilized twigs of many forest trees, on roots, and on bean plugs, carrots, potatoes, sweet potatoes, bread, corn meal, oatmeal, rice, sugar solutions, bouillon, various synthetic media, and a large number of agar media. In fact, in the saprophytic condition the fungus seems to be almost omnivorous.

Isolation

The fungus is most readily isolated by removing, under sterile conditions, a small piece of the diseased tissue of the inner bark, especially in the

youngest part of the canker, and transferring it to agar tubes. Anderson and Anderson (1913) have described in detail various other methods of isolation. Either kind of spores may be sown on the agar, or streaks may be made on agar slants with the spore horns, or the ascospores may be permitted to fall on agar plates after natural ejection.

General cultural characters

There are certain characters that are common to the growth of the organism on most of the artificial media commonly used, particularly the agars.

Murrill (1906 a: 146) says: "When grown in artificial culture, the mycelium of the fungus is at first pure white, changing to yellow with age, and the fruiting pustules are a beautiful yellow." He finds that the fruiting pustules are produced in eleven days, and mature spores in the process of discharge in twenty days. Neither Murrill nor any writer since 1906 has succeeded in producing the ascospore stage in culture.

Clinton (1909: 886) describes the same color change on lima-bean agar, and adds: "The threads form a rather hard crust on the surface of the medium, and in this the Cytospora fruiting stage develops as numerous small elevations. The spores, after maturity, ooze out on the pustules as lemon-yellow drops, which later become light chestnut-brown in color."

Anderson and Anderson (1912 a) advocated the cultural characters as a means of distinguishing this species from the other very closely related and confusing species of Endothia. Since then, Pantanelli (1912), Clinton (1913), and Shear and Stevens (1913 a) have studied this phase, and they all find additional cultural characters on a number of media by which these forms may be distinguished. It is not our purpose to discuss these differences in this publication, since they do not directly concern us at this time. Only the most important characters that relate to *E. parasitica* will be mentioned. For a more complete discussion the reader is referred to Shear and Stevens (1913 a), where the cultural characters on a large number of media are described in detail.

Sterilized twigs

On twigs of all the common forest trees that were tried (Anderson and Anderson, 1912 a), the fungus produces a short, white, web-like growth over the surface of the twig, with heavier bunches of mycelium which later become orange-colored, where the pycnidia are to develop. On the cut ends of the twigs there is developed a thick, felt-like, orange mycelial growth, but this never extends out on the bark as in *E. radicalis*. The same characters were found by Shear and Stevens (1913 a), except that

these investigators call the color of the mycelium on the cut surface "cadmium yellow."

Potato agar slants

When streak cultures are made on potato agar slants with spore horns, the mycelium begins along the streak as a white weft and spreads rapidly toward the edge. In four days, at ordinary room temperature, the orange color begins to appear along the streak, and it broadens as the mycelium grows out until the whole surface of the slant is covered with a deep orange growth. This test was used most extensively by the Andersons in distinguishing this species from the other Endothiæ. Shear and Stevens (1913 a:12) record the same phenomenon on this agar, and add: "Within six days, the mycelium, especially at the base of the agar slant took on a peculiar granular metallic appearance. . . . This portion of the culture was light orange yellow by reflected light and orange by transmitted light. The peculiar surface appearance might perhaps be called 'brassy.' This metallic appearance has been found to be the most constant and reliable distinguishing character of *E. parasitica* on potato agar. In twelve to fourteen days small pycnidial pustules appeared in the upper portion of the tubes, and the agar just below the mycelium became warbler green, changing later to olive green."

Often potato agar cultures after three or four months turn to a deep purple or wine-color. H. W. Anderson (Anderson, P. J., 1914) found that this purple color is due to the fact that the long growth of the organism on the agar causes the latter to lose its acid character and become alkalin. The yellow pigment (aurine) in the mycelium goes into solution in an alkalin medium and turns purple. The pigment then escapes from the cells and is suffused throughout the agar.

Relation of light

The fungus grows just as rapidly in darkness as in light, and also produces the yellow pigment but not so abundantly. D. C. Babcock, working with one of the writers of this bulletin, found, however, that in total darkness no pycnidia are produced, but just as soon as the culture is brought into light the pycnidia begin to form. This fact accounts for the concentric rings of pycnidia so noticeable on plate cultures. One circle is produced each day. Doctor Shear, in a letter to the writers, states that pycnidia are produced in darkness.

Relation of temperature

Shear and Stevens (1913 a:9) studied the relation of temperature to the growth of the mycelium in pure cultures. Their results are thus

summed up: " From these records it will be noted that the minimum temperature for all was 9° C. and that all failed to grow at 7° C. The maximum temperature for *Endothia parasitica* . . . was 35° C. . . . The optimum appears to be near ordinary room temperature, that is, about 22° to 24° C."

Tannic acid

Since the chestnut tree has a relatively high tannic-acid content, the question naturally arises as to the relation of tannic acid to the growth of the fungus. Clinton (1913:407) grew the fungus on cultures containing various amounts of tannin, and summarizes his results thus: " (1) Both fungi [referring to *E. parasitica* and *E. gyrosa*] can use tannic acid, at least in small amounts, as food — shown by the blackening of media through oxidation, loss of acidity, more luxuriant growth, with a low per cent of the acid added, than without it, and a slight growth on agar-agar with tannic acid as the available source of food. (2) Higher percentages of tannic acid (four per cent and above) are detrimental to a vigorous growth of either of these fungi, and finally (10 to 14 per cent) entirely inhibit their growth. But with the true blight the tolerance is apparently greater by 2 to 4 per cent than that of the saprophytic *E. gyrosa*. (3) Long-continued cultivation of the parasitic variety in artificial cultures without tannic acid probably lowers its tolerance to the higher percentages of tannic acid. (4) Gradually passing these fungi in cultures from the lower to the higher percentages of tannic acid apparently raises their tolerance to it." Clinton suggests that the tolerance of a higher percentage of tannin probably bears some relation to the parasitism of *E. parasitica*.

ECOLOGIC RELATIONS

Murrill (1906 a:153) gives his opinion (1) that winter injury possibly accounted for the epiphytotic, (2) that there was a decline in constitutional vigor, because of coppicing, which predisposed the tree to attack, and (3) that cultural conditions could determine relative resistance to attack. The effect of these ecologic conditions, as well as of drought, in increasing the susceptibility of the tree is upheld by Clinton (1909:887–889; 1911: 717; 1913:389–407). In the last publication Clinton (1913:390–391) writes:

" Now, if the condition of the host bears no relation to the rise and spread of the disease, the writer knows of no satisfactory explanation for its sudden and destructive appearance in this country except its importation from some foreign country. The evidence to date, however, is very strongly against the idea that it is an imported pest, as we shall show later. Among the farmers in Connecticut who have been able to watch this disease rather closely there are many who believe that the weakened vitality

of the chestnuts has had considerable to do with its development and spread in this State. The writer more than any one else has advocated this view, and we propose to give here the reasons we have for holding it. Briefly expressed, they are as follows:

"The chestnut blight was brought to sudden prominence just after the severe winter of 1903–1904, which injured and killed fruit and forest trees in general along the coast and watercourses, of which New York City was the central point. The resulting enfeebled condition of the chestnut enabled the blight, a previously inconspicuous parasite, to spring into sudden prominence on these trees and to gain credit for the death of others which had been largely or entirely due to winter injury. Since then we have had one or two severe winters, and more especially several dry summers, that have injured not only the chestnut, but other forest trees over an extended area. Due to its successful attack on the weakened trees, the blight fungus has perhaps acquired an added virulence that has enabled it to attack apparently healthy trees, especially those of sprout renewal. The enfeebled condition of the chestnut trees and their consequent susceptibility to the blight may possibly be related to some lessened chemical activity in the bark and newly-formed wood, such as the production of tannic acid, for instance. If so, then when this has returned to its normal production through favorable weather conditions, the blight should gradually become correspondingly less aggressive."

Opposed to these views we have those of Metcalf and Collins (1910): "A debilitated tree is no more subject to attack than a healthy one. [See also Metcalf, 1910.] Dry weather checks the disease by suppressing spore production. . . . Winter injury is not common over the whole range of the bark disease, but may be locally important in producing lesions through which the parasite enters. Winter injury bears no other relation to the bark disease." Metcalf (1912 a:225) writes: "No definite evidence, experimental or otherwise, has been adduced to show that a tree with reduced vitality is more susceptible to infection, or that the disease spreads more rapidly in such a tree than in a perfectly healthy and well-nourished tree of either seedling or coppice growth, provided that such reduced vitality does not result in or is not accompanied by bark injuries through which spores can gain entrance." (See also Metcalf, 1912 b: 79–82.) The writers think that in the above quotation Metcalf has well summed up the apparent fallacies in the preceding statements made by Clinton.

By observations the writers have found no connection between any ecologic conditions and any variation in the susceptibility of the tree. However, ecologic conditions may determine the rapidity of spread of the fungus through the infection courts opened up, such as winter- and

drought-injured bark and limbs, hail, and other mechanical injuries, and the like.

The writers have proved, through thousands of inoculations made in the States of Pennsylvania and New York, that the healthiest trees are entirely susceptible. Winter-injured trees, even if dead, could not be more susceptible.

Anderson and Babcock (1913) were unable to find any conditions under which one tree is more susceptible than another. One of the writers of this bulletin (Rankin, 1914) has shown by a careful series of inoculation and growth studies, along with accurate determinations of the water and air content of the bark, that in 1912, at least, no connection between drought and change in susceptibility was found. The majority of the inoculation work reported in the same paper was performed on coppice growth which had grown from trees cut about five years previously. These trees had not, therefore, been subject to the severe winters which Clinton has claimed, predisposed the chestnut. In no way were the results of inoculations made on this five-years-old coppice any different from those made on older trees. In cutting several trees near Napanoch, a few were found that showed a dead area which may have been produced by the severe winter of 1903–1904. In one case the injured area had decayed, leaving a ring-shake. In other trees no injury was apparent. No difference in susceptibility, however, was noted.

The writers would summarize their belief, based on observation and experiment, that, without regard to any ecologic relations, the native chestnut is entirely susceptible because of the parasitic potentialities of the fungus, which have not heretofore had an opportunity to be demonstrated.

CONTROL

METHODS THAT HAVE BEEN ADVOCATED

Merkel (1906: 101–103) attempted to control the disease in the New York Zoölogical Park, where he first discovered it. In the summer and fall of 1905 he had all the diseased limbs removed from four hundred and thirty-eight trees, and the trees were then sprayed with bordeaux mixture. Only one application was made. No further report appeared on this experiment except the statement of Murrill (1906 b: 205): "An attempt was made by Mr. Merkel, chief forester at the Zoölogical Park, to control the disease by spraying, but I believe he considers the condition quite hopeless. Practically all of the chestnut trees within his jurisdiction appear to be dying rapidly."

Murrill (1906 a: 152–153) was the first to give advice on control, his advice being as follows:

" The treatment of a disease of this nature must, of course, be almost entirely preventive. When once allowed to enter, it cannot be reached by poisons applied externally, nor can the spores, which issue continuously and abundantly through eruptions in the bark be rendered innocuous by any coating applied at intervals. On the other hand, no poisonous wash, even though covering every part of the tree, can prevent the germination of the disseminated spores when they fall into a wound, since the wound opens up fresh tissues unprotected by the poison.

" The spraying of young trees with copper sulfate solution, or strong bordeaux mixture, in the spring before the buds open might be of advantage in killing the spores that have found lodgment among the branches during the winter, but the real efficacy of this treatment is so doubtful that it could not be recommended for large trees, where the practical difficulties and expense of applying it are much increased. Nursery trees should be pruned of all affected branches as soon as they are discovered, and the wounds carefully dressed with tar or paint or other suitable substance. Vigilance and care should largely control the disease among young trees. With older trees all dead and infected wood should be cut out and burned and all wounds covered without delay."

Murrill (1906 b:209) says also: " I have no treatment to suggest further than the preventive measures already mentioned." In 1908 he adds (1908 a:24–25): " Preventive measures have apparently not affected it in the slightest degree. The pruning of diseased branches has entirely failed to check it, even in the case of very young trees. Branches have been carefully removed and wounds covered, leaving the tree apparently entirely sound, but upon inspection a few weeks or a few months later they would be found badly diseased at other points." In the same publication (page 30) he advises eradication measures for spot infections in the woods: " Owners of standing chestnut timber within the affected area are advised to cut and use all trees, both old and young, that stand within half a mile of diseased trees, unless protected from infection by wind-blown spores, by dense forest growth, or some other natural barrier." Hodson (1908:7) advises: " (1) To cut out the diseased trees, (2) to institute a quarantine against the shipment of infected material."

Metcalf (1908 b:490) also advocated the cutting-out method for limiting the spread of the disease. He writes: " In certain localities where the disease is just appearing it would undoubtedly be possible, by prompt cutting down or treatment of all infected trees and by very careful inspection, to maintain a zone free from the disease, and hence keep the disease out of the still uninfected country beyond." Clinton (1909:890) writes: " No efficacious treatment for the prevention of the trouble has yet been found, though spraying, pruning, and burning of infected trees have been advocated."

Metcalf and Collins (1909:49–52) take up in detail the means of control of the disease. After stating that the distribution of the fungus to distant points would be accomplished largely by the shipment of diseased nursery stock, they point out the necessity of rigid inspection of nursery stock shipped into uninvaded territory. They also advise a campaign of education in order to acquaint the public with the nature and appearance of the disease. They then point out the necessity of promptly cutting and burning newly affected trees so that spot infections may be arrested. They say: "Almost the only treatment that can at present be safely recommended as surely retarding the spread of the disease to a greater or less extent is one which will never be of practical use except in the case of orchard trees or certain valuable ornamental trees. It consists essentially in cutting out the infected branches or areas of bark and carefully protecting the cut surfaces from outside infection by means of a coat of paint or tar. This cutting must be thoroughly done and the bark of every infected place entirely removed for a distance of at least an inch (where the size of the branch permits) beyond the characteristic, often fan-shaped, discolored areas produced by the growing fungus in the inner bark. All small infected twigs or branches should be cut from the tree, the cut being made well back of the diseased area. A pruning knife with an incurved tip, a hollow gouge, or any other clean-cutting instrument will serve for cutting out diseased spots. . . . The paint or tar may be applied by means of a good-sized brush, care being taken to cover every part of the cutting." (See Figs. 96 and 97.)

It remained for Metcalf and Collins (1911:10–24) to suggest definite methods for arresting the country-wide progress of the disease. They outlined their method of scouting and cutting-out in detail (pages 12 to 17 of reference cited). The procedure is summarized thus (page 24):

"The only known practical way of controlling the disease in the forest is to locate and destroy the advance infections as soon as possible after they appear and, if the disease is well established near by, to separate the area of complete infection from the comparatively uninfected area by an immune zone. Advance infections should be located by trained observers and destroyed by cutting and burning. As the disease develops almost entirely in the bark, this must be completely destroyed (burned).

"In order to carry out the above methods it is essential that the several States concerned secure necessary legislation and appropriations, following the example of Pennsylvania, as no law exists whereby the Federal Government can undertake such work and cooperation among private owners without state supervision is impracticable."

The method proposed led to much discussion among pathologists. There were a few who openly criticized the method as impractical, and

pronounced it a possible source of wasteful expenditure of state money. Others were more neutral and awaited additional evidence. The majority, however, were of the opinion that further research must be made before the practicability of any method could be actually proved. Stewart (1912) gave many reasons for not indorsing the method. He said: (1) the method was not supported by experimental evidence; (2) " no such method of controlling a fungous disease has ever been attempted "; (3) known facts concerning the disease did not make the method appear feasible; and (4) "it is better to attempt nothing than to waste a large amount of public money on a method of control which there is every reason to believe cannot succeed."

FIG. 96.— *Method of cutting out a canker on the trunk. The wood is cut out an inch below the bark*

Murrill (1912) thus presents his views as to why the chestnut canker cannot be controlled by the cutting-out method:

" 1. It is impossible to locate all advance infections, these not being apparent even under close inspection.

" 2. It is practically impossible to cut down and burn all infected trees after their discovery.

" 3. Even if these trees are cut, it is impossible to discover and eradicate the numerous infections originating from millions of spores produced on these trees and distributed by birds, insects, squirrels, wind, and rain.

FIG. 97.—*Trunk from which four cankers have been cut out. After sterilization the wounds are completely coated with a wound dressing*

" 4. Even if it were possible to cut and burn all affected trees, for ten to twenty years afterwards numbers of sprouts would grow up from the

roots of these trees and continue to die from the disease and to spread the infection.

" 5. Supposing that it might be possible to eradicate all advance infections, what method is proposed that is at all feasible for combating the disease in its main line of advance? All of the foresters connected with the United States Government and the entire army of the United States would be utterly powerless to oppose its progress.

" 6. Although the chestnut canker has been known and experimented with since 1905, there is not a single instance where an individual tree or a grove of trees affected by the disease has been saved. If it is impossible to combat the canker under the most favorable circumstances, how would it be possible to succeed with an extensive forest? The published account of the extermination of the chestnut canker in the vicinity of Washington, D. C., cannot be relied upon. The trees most conspicuously affected there have been cut and burned, so that the presence of the disease is not readily apparent, but with each season additional trees will be affected and the attempt to stay the disease will be abandoned, especially when the main line of advance, which is now in northern Maryland, reaches the Potomac River."

Clinton (1912 c:82) says that in the main he agrees with Stewart and Murrill.

Some months previous to this discussion the State of Pennsylvania had appropriated a large sum of money for the investigation and control of the chestnut-tree blight disease. Metcalf and Collins (1911:14–17) quote this act, which embodies the scouting and cutting-out method described in the same publication.

During the summer of 1911 Doctor Metcalf appointed several agents who scouted in a preliminary manner the different States concerned in the control of the disease. With the information thus available (Metcalf and Collins, 1911:6), the authorities of the different States considered means of controlling the disease. Those States north of Pennsylvania took no definite action, mainly for two reasons: (1) the cutting-out method (the only method proposed) was not sanctioned by some persons to whom the state authorities looked for advice; (2) the disease had gained such a foothold that its economic control by the cutting-out method could hardly be expected by the most radical supporters of the method.

The published reports on field operations for the control of the disease in Pennsylvania are limited to the reports for the period from July 1 to December 31, 1912. The work of organization and preliminary scouting occupied the interval from the time of the appointment of the Commission in the summer of 1911 until the spring of 1912. The Commission reports (Penn. Chestnut Tree Blight Com., 1913:11):

"From the beginning, a more or less definite division has been maintained between the slightly infected western portion of the State and the badly infected eastern portion, these divisions being called the Western and Eastern Districts, respectively. In the two districts quite different restrictions are maintained with respect to the method of procedure in handling diseased trees. The line of demarcation between these districts, as at present understood, is the eastern boundary lines of Fulton, Huntingdon, Mifflin, Center, Clinton, Lycoming, Sullivan, and Bradford Counties. . . . The field work [page 21] west of the advance line has for its object primarily the total eradication of the blight, and the checking of further westward spread. East of the advance line where the bulk of the chestnut trees is located, it is the duty of the Commission to acquaint owners of timber with the facts relating to the blight. . . . at least in time to cut out the diseased trees before they deteriorate in commercial value. . . . In each district a district superintendent has been appointed to direct the field work. . . . The western district was subdivided into seven divisions of five to seven counties each, and five divisions were made in the eastern district. Each division has been in charge of a Supervisor. A field agent was detailed to conduct the work in a county and as many scouts as necessary were assigned him as assistants. . . . When the examination [page 23] of each tract was completed a data card, giving all the necessary information relative to the tract, was sent to Field Headquarters . . . The plan [page 25] now being followed when a spot infection is found is to blaze the infected trees at breast height and also at the base . . . and the infected trees numbered consecutively. . . . The points [pages 26–27] to be emphasized in eradicating spot infections are:

"1. Take all possible care to prevent injuries to surrounding chestnut trees and sprouts in felling the infected tree. If it is necessary to clear away brush to facilitate cleaning up after felling, any small chestnut sprouts should be cut flush with the ground. Experience has shown that such stubs often become infected if near a diseased tree.

"2. Cut all stumps as low as possible, to lessen expense of peeling and to save merchantable timber in the log.

"3. Destroy all diseased portions of the tree showing pustules, by burning on the spot, immediately, either the bark or entire sections of the tree which show cankerous areas.

"4. Either utilize all unbarked portions of infected trees within a brief time after they are cut, or, if it is desired to permit this material to remain in the vicinity of healthy chestnut trees, peel the bark from all portions of the trees which it is desired to retain.

"5. In every case peel the bark clean from the stumps to an inch or

two below the surface of the soil. Experience has shown that the stumps of infected trees and portions of the green tops which are permitted to lie for several months on the ground are almost certain to become infected if the bark is permitted to remain on them, even though no cankers exist on the stump at the time the tree is cut. Some of the largest spots of infection have developed from unpeeled stumps. The spores germinate on the sappy surface of the stump and the mycelium grows downward through the cambium, and in the course of a year or two reaches the sprouts which come around the base of the stump. Little infection in the sprouts is found where the stumps have been carefully peeled. Furthermore, the sprouts have more vigor and are better rooted when they come from peeled stumps, since in this case they must start from beneath the soil and can so form their own roots." (Figs. 98 to 101.)

FIG. 98.— *Peeling the base of a diseased tree. Woodsmen find this easier than peeling the stump after the tree is cut*

As to the results of the cutting-out method, the following is quoted from the same report (page 27): "Sufficient time has not elapsed since the Commission began work to determine the efficiency of sanitation in checking the disease. . . . Forty-two tracts on which the original infection was cut out during the early part of 1912 were reinspected during November and December of this year (1912). The number of diseased trees in these spots prior to cutting ranged from a single tree to ninety-three, the total number of diseased trees on the forty-two spots being five hundred

and fifty-six. On reinspection, twenty-eight out of the forty-two spots showed no recurrence of the blight; in three cases a single new infection was found, and in six cases there were two recently infected trees. The highest number of new infections numbered thirteen trees. In the forty-two spots averaging 13.25 original infected trees each, one hundred and fifty-six reinfections occurred, or 3.7 infections per spot. In two thirds of the forty-two spots no blight reappeared, and the new infections which developed in the remainder equaled only two sevenths of the number of trees originally diseased. These spots were located in the region of very slight infection in Elk, Clearfield, Center, and Fulton counties."

FIG. 99.— *Cutting down the tree that has been peeled at the base*

The results of the oldest experiment that is reported are as follows (pages 32–33 of the same report):

"In order to get information concerning the effectiveness of two different methods of cutting out diseased chestnut, a stump-to-stump count of 100 stumps each was made in November, 1912, on two different tracts located at Haverford. In one of the woodlots the infected trees were cut in the fall of 1910, and the stumps peeled, and all brush destroyed by burning, but the burning was not done over the stumps. On this tract a hundred stumps had 1354 vigorous

sprouts, on 254 of which the blight was present. In other words, 82 per cent of these sprouts were free from disease, and of the infected sprouts, 99, or 39 per cent, were infected at the base mostly from diseased bark left on the stump.

"The second tract, used for comparison with this, is located about one half mile distant from the first tract and was cut about the same time. The brush was burned and all the merchantable wood used, but the stumps were not peeled. As near as could be determined, the two woodlots received identical treatment except that the stumps were peeled in one case while they were left with the bark on in the other. On this

FIG. 100.— *Peeling the trees that had cankers*

tract the 100 stumps had 1406 vigorous sprouts, 1115, or 79.3 per cent of which were infected, 22.2 of the infections were basal."

The Pennsylvania Commission, after having started these extensive state-wide experiments in control, passed out of existence in July, 1913, through failure of the Legislature to appropriate sufficient funds with which to continue the work as it had been begun. Much that was valuable had been accomplished in scientific research, but the field experiments in cutting-out, on which the greater part of the $275,000 was spent, were only begun and can in no wise be of more than very temporary benefit to the State.

In New York State the survey made by one of the writers of this bulletin proved that the disease had a greater stronghold than had been supposed. With the vast area that was then affected, the problem of control

presented many difficulties. The chestnut in the part of the State west of the line of advance (Fig. 78) was of considerable value and worthy of being saved if this had been economically possible. The region that contained the greatest amount of the valuable chestnut timber of the State, however, lay in the hopelessly affected area. One of the writers, in making a report to Doctor Metcalf in October, 1911, made the following statements: "It is well, however, to consider the difficulties involved. The advancing line [Fig. 78] is in a region in which the topography is extremely rough, the hills are thickly wooded, and the chestnut is

FIG. 101.— *Burning the branches over the stumps. All broken-off branches and chips are raked up and burned*

abundant. Under such adverse conditions, it would be, to say the least, a gigantic task if not indeed an impossible one. If natural barriers were more favorable, such a scheme might be more feasible." No special appropriation or plan to control the disease was made. The position of State Forest Pathologist was inaugurated in July, 1912, and one of the writers was appointed to this position. Field investigations on the chestnut canker were conducted until October, 1913. These are reported elsewhere (Rankin, 1914).

The writers understand that West Virginia and Virginia (Gravatt, 1914) are scouting for spot infections and eradicating them as fast as

possible. No elaborate organization has been attempted and no large sums of money have been appropriated for these operations. The regular authorities are in charge of the work. The Office of Forest Pathology at Washington is still maintaining research work on the disease and has employed S. B. Detwiler, formerly in charge of the field operations in Pennsylvania, to check the results of the cutting-out done in western Pennsylvania. The report on this work is soon to be published (Detwiler, 1914). North of Maryland no attempt is being made to control the disease.

GENERAL DISCUSSION OF CONTROL MEASURES

Exclusion measures

In regions still unaffected the consideration of exclusion measures is highly important. Diseased nursery stock has been proved the most dangerous means of introduction of the fungus into new localities. The original spot infections in this country were, without much doubt, started from diseased nursery trees shipped from the Orient. Five out of nine of the spot infections in western Pennsylvania were traced to diseased nursery stock. The only spot infection known in western New York — at Penn Yan — was in an orchard.

In order to prevent the shipment of diseased stock the trees should be inspected when they are packed and again when they reach their destination. A blanket certificate issued yearly to nurseries is worthless in the case of this disease, as with many others. As a part of the control measures instituted by the Pennsylvania Commission, all nursery trees were inspected and tagged individually before packing. The inspection of trees at their destination is not always sure to exclude diseased trees, for incipient infections are likely to be overlooked.

Because of the uncertainty of inspection of shipments and the extreme virulence of the parasite, it seems highly desirable that an absolute quarantine be placed on the affected region of the eastern United States, which would prevent the shipment of chestnut stock outside the prescribed limits. This would protect outlying or distant parts of our own country and would aid foreign countries in excluding the disease. It is especially incumbent on foreign countries that have chestnut to protect, to take the most rigid measures in order to prevent this disease from gaining a foothold.

The fact that diseased nursery stock is not the only means of transporting the fungus from an affected region must not be overlooked. Parts of diseased trees barked or unbarked may serve to carry the fungus. The following points show the danger there is in transporting unpeeled

logs or tree parts outside the affected regions: (1) ascospores will germinate after having been kept dry for two and one half years; (2) conidia will germinate after having been kept dry for at least a year; (3) mycelium retains its vitality after having been kept dry for at least ten months; (4) perithecia, after having been kept dry for seven months, will again eject spores on being moistened.

Peeled timber may serve to carry the fungus, for the mycelium, which penetrates at times to the fifth annular ring of the wood, will under moist conditions produce superficial pycnidia and spore horns on the surface of the wood.

There are chances, of course, that even a strict quarantine on nursery stock or chestnut products will not eliminate all the means of introduction into a new territory. The fungus, in the form of either mycelium or spores, may be carried on or in other plant parts or by birds, wind, or other uncontrollable agents.

Eradication measures

There are two kinds of conditions under which eradication measures may be carried out, namely, arboricultural conditions and forestry conditions. In the case of orchard and ornamental trees the principles of sanitation and tree surgery have been advanced for the control of the canker. But even as early as 1908 Murrill says these means are without avail. Vast sums of money were expended by individuals in order to save such trees in cities and on private grounds in the regions first affected. Failure attended all attempts. This was largely due to four reasons: (1) the work was done by commercial tree surgeons who had had no special training; (2) it is impossible to find and remove all affected parts of a diseased tree, especially incipient infections and cankers under rough bark; (3) reinfection is constantly possible; and (4), a reason not the least in the majority of cases, the fact that the mycelium entered deeply into the wood was ignored, and the cankers renewed growth by the mycelium growing from the wood back into the bark. The last-named fact has been brought out by experiments conducted by Collins (1912 a) and by members of the staff of the Pennsylvania Commission (1912 b:2).

The removal of dead and dying trees and of all refuse from the surgical operations is necessary. Also all wounds must be protected immediately by an enduring covering (Fig. 97, page 598). Ample suggestions in regard to surgical methods are given by Metcalf and Collins (1911:18–20), Collins (1912 a), Pennsylvania Chestnut Tree Blight Commission (1912 b), and others. A later report (Pennsylvania Chestnut Tree

Blight Commission, 1914) says that careful tree-surgery work will often save affected trees.

With regard to the eradication measures to be applied in the forest, the cutting-out method of Metcalf and Collins (1911:10–17) embodies the principles mentioned above. The method, although put into practice in Pennsylvania in 1912 and 1913, will not now, it seems, be demonstrated, since all concerted attempt at country-wide control has been abandoned. At the present time, however, we can theorize on the practicability of the method with more certainty than was possible at the time of its original consideration in 1911.

The writers believe that the method would not have succeeded economically even if it had been vigorously prosecuted from the time of its proposal. In addition to the reasons given by Stewart (1912) and Murrill (1912) which in their opinion make the method impossible, later research has constantly added evidence of the important part played by numerous disseminating agents that must be reckoned with. Careful surveys and the elimination of all spot infections outside the affected region would accomplish temporary control. But even with a carefully executed quarantine on nursery stock and chestnut products, the chances of reinfection from a distance or reappearance at an old spot infection would necessitate constant resurveys, which, if thorough, would be expensive and could not even then be expected to absolutely eradicate the disease. The principal difficulty, however, would be the main line of advance, where on the one side nothing would be done and immediately adjacent complete control would be attempted by eradication. The immune zone suggested in this scheme is the only way of meeting this. Just how wide such a zone would have to be in order to obviate infection due to wind dissemination of ascospores would vary with conditions. The chestnut-free belt in the Catskill Mountains, varying from thirty to forty miles in width, has apparently furnished protection for the area west of the mountains. The mere felling and utilization of all the trees, however, in a zone twenty miles around the affected area as it was in 1911, would be an enormous task, consuming much time and money. Taking into consideration the work and the money required in order to put the whole scheme into operation, it does not seem logical to believe that the chances of success warrant the risk.

Hope is expressed in a publication by Craighead (1912), who states that certain insects found eating the perithecial pustules of the canker fungus may aid in eradicating the fungus. Ruggles (Penn. Chestnut Tree Blight Com., 1914), however, writes: "The particular insect (*Leptostylus maculata*), carrying the 336,900 spores mentioned is one of the beetles named in a recent press notice of the United States Depart-

ment of Agriculture, as being very active in eating spores of the blight fungus. Therefore this beetle while destroying spores of the blight is at the same time covering its body with thousands of other chestnut blight spores which it carries from tree to tree, making it probably an injurious insect instead of a beneficial one in this respect."

Protection measures

The only experiments reported on spraying were those of Merkel and the Pennsylvania Commission. The single application of bordeaux mixture made at the New York Zoological Park by Merkel in 1905 failed to check the disease. The Pennsylvania Commission conducted spraying experiments at Kenneth Square, Pennsylvania. Bordeaux mixture 4–4–50 was applied with a power sprayer every two weeks from April until the middle of November, 1912. No definite results are reported from this experiment (Penn. Chestnut Tree Blight Com., 1913:51, Figs. 53–55).

Immunization measures

Many writers have suggested the possibility of selecting and breeding immune varieties. Morris (1914) sums up observations on different varieties planted among affected American chestnuts. Van Fleet (1914) has found Asiatic chinquapin hybrids promising. With the relatively high degree of resistance shown by the Japanese and Chinese chestnuts, there may be a possibility of breeding within these varieties. Crosses with the American chestnut seem hopeless. Metcalf and Collins (1911:20) write: "If the seed [of Japanese chestnuts] is raised in America, the trees are more than likely to be hybrids with the American chestnut and to vary greatly in resistance to the bark disease."

The investigators of the Pennsylvania Commission applied many kinds of fertilizers around trees and carefully recorded the effect on the rate of growth of cankers. At Mount Gretna, Pennsylvania, the following substances were used in different combinations and quantities: burned lime, nitrate of soda, acid phosphate, muriate of potash, and kainit. At Emilie, Ambler, and Valley Forge, Pennsylvania, iron filings and coal and wood ashes, in addition to the substances mentioned above, were applied. At Mount Gretna the trees were old and thick-barked. The cankers were outlined in spring at the time of application, and again in late fall. When the measurements were compared with the checks no differences were found except within the limits of the usual variations. In the experiments at Emilie, Ambler, and Valley Forge no variation on rate of growth of cankers was obtained.

Also, at Emilie many secret preparations, applied as fertilizers or as injections, were tried. These were methods advocated by various laymen

who considered they had a specific for the control of the canker. One of the writers of this bulletin served as a member of the Board of Review (Penn. Chestnut Tree Blight Com., 1914) which passed on the records of these tests as well as on those of the fertilizer tests. The report of the board stated that not one of the treatments had affected in the least the rate of growth of the cankers. In a large number of cases, only single very small cankers were present on the trees at the time of treatment, so that the tests were fair.

Many methods of tree medication have been brought forth by quacks since the chestnut canker began its ravages. The Pennsylvania Commission furnished trees for testing these methods to all who applied for a test to be made of their specific. As mentioned above, these all failed to produce the desired effect.

Dr. C. Rumbold (Penn. Chestnut Tree Blight Com., 1913:45–47) has been working on the effect of different substances injected into the chestnut, but as yet has reported no successful results.

RECOMMENDATIONS FOR TIMBER OWNERS IN NEW YORK STATE

The warning may well be repeated against planting chestnut anywhere for orchard or ornamental purposes. The speedy utilization of all diseased trees may serve as a local temporary control measure if carefully carried out. However, as it is now apparent that little can be done to control the disease, the consideration of the best means of utilizing the present stand is important to the timber owner (Nellis, 1914).

Three separate conditions exist in New York State at present: (1) in the Hudson River valley, conditions exist varying from total destruction to more or less numerous spot infections; (2) in the Roundout and Delaware River valleys, spot infections are numerous and probably but little damage has been done to the trees as a crop; (3) west of the Delaware River valley, spot infections are few and of small extent. It is important, then, to consider what can be done in these different sections in order to minimize the damage.

In the Hudson River valley it is a question of speedy utilization of dead and dying trees for the most part. This is a necessity for two reasons: (1) in order to obtain profit for the owner which will otherwise be decreased, and (2) in order to rid the section of this disease- and insect-breeding material. The owner will undoubtedly find some more or less profitable market for the greater part of the accessible timber now going to waste, if he makes a study of his local conditions. He may also obtain information from the federal and state departments. The importance of ridding the country of the dead material, which will otherwise become a breeding-place for other fungi and insects, cannot be overestimated.

In the Delaware and Roundout River valleys it is a question of utilization of immense quantities of second-growth chestnut of small dimensions. It seems that the work in this region, as well as in the Hudson River valley, is of a magnitude sufficient to warrant a careful study by the proper state officials in order that owners may receive helpful advice on conditions, markets, and methods. This would be an immediate phase of conservation worth while to the State of New York.

In the central and western parts of the State a careful watch should be kept for new spot infections, and the diseased trees eradicated as soon as discovered. Plans for future replacement of chestnut by other species should be made now, even before the chestnut dies, so that a forest may be growing up in the meantime.

One important factor to be considered at present through the whole State, and especially the eastern part, is what will become of the land that previously has grown chestnut. Careful study should be made of silvicultural conditions in these regions, followed by an attempt to obtain a stand of a desirable species that will replace the chestnut. Otherwise the tree weeds and other factors will interfere and will increase indirectly the damage done by the canker.

THE OUTLOOK

At present we know of nothing that will prevent the extermination of the American chestnut tree. Every measure of control that has been tried has been abandoned north of West Virginia and the Potomac River. Some persons have expressed the belief that nature herself will intervene to prevent destruction of the species; the virulence of the pathogen will abate, the resistance of the host will be increased, or natural enemies — insects or fungous parasites — will destroy, or at least check, the pathogen. Up to the present, however, there has been no indication of relief along any of these lines. But we do not believe that the ingenuity of our scientists has been exhausted; that further research will bring to light some method of combating the disease is not beyond the limit of probability. Such research not only should be continued, but also should be augmented; even if effective control measures will not be evolved until it is too late to save the present stand of chestnut, they will be of service in combating other forest epidemics which will undoubtedly come in the future.

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1912 a The chestnut tree canker disease. Phytopath. **2**:99.
Abstract of paper read before the American Phytopathological Society, December, 1911. Reports the forcible discharge of ascospores, and the presence of the superficial pycnidia on wood.

1912 b How further research may increase the efficiency of the control of the chestnut bark disease. Pennsylvania Chestnut Blight Conference. Stenographic rept., p. 46–48. Harrisburg, 1912.
Calls attention to the importance of wind-blown ascospores; reports the closely related saprophytic form in Virginia and Pennsylvania.

1913 Some field experiments with the chestnut canker fungus. Phytopath. **3**:73.
Abstract of paper read before the American Phytopathological Society, January, 1913. Inoculation experiments. Author finds that drought does not alter susceptibility of the tree.

1914 Field studies on the Endothia canker of chestnuts in New York State. Phytopath. **4**:—. (In press.)
Records field experiments with regard to the life history of the fungus and its distribution in New York State.

Rehm, H.

1907 Ascomycetes exs. Fasc. 39. Ann. myc. **5**:210.
Regards the chestnut fungus as one of the Hypocreaceae, and proposes the combination *Valsonectria parasitica* (Murrill) Rehm.

Ruggles, A. G.

1913 Notes on a chestnut-tree insect. Science **38**:852.
Discusses life history of the bark insect commonly known as the bast-miner and previously erroneously referred to by Metcalf and Collins (1911) as *Agrilus bilineatus;* believes this insect has an important bearing on the spread of *Endothia parasitica.*

Shear, C. L.

1912 a The chestnut bark fungus, Diaporthe parasitica. Phytopath. **2**:88–89.
Reports that *Endothia radicalis* (Schw.) and *Diaporthe parasitica* Murr. are distinct.

1912 b The chestnut blight fungus. Phytopath. **2**:211–212.
States that the European *Endothia radicalis* is identical with *Diaporthe parasitica* Murr., and that it was probably introduced into America from Europe.

1913 a Endothia radicalis (Schw.). Phytopath. **3**:61.
Author retracts his former statement, and now believes that *E. radicalis* (Schw.) is not the linear-spored form but the narrowly-oval-spored form, *E. virginiana* Anders.; does not now believe that the blight fungus was introduced from Europe.

1913 b The type of Sphaeria radicalis Schw. Phytopath. **3**:191–192.
Examines Fries's type specimen of *S. radicalis,* and decides that it is identical with the form called *E. gyrosa* by Clinton and *E. virginiana* by the Andersons.

Shear, C. L., and Stevens, N. E.

1913 a Cultural characters of the chestnut-blight fungus and its near relatives. U. S. Plant Indus. Bur. Circ. 131:3–18.
Describe in detail cultural studies of all the American species of Endothia, and consider *E. parasitica* as a distinct species.

1913 b The chestnut-blight parasite (Endothia parasitica) from China. Science **38**:295–297.
Give cultural and inoculation experiments proving that the fungus collected in China is identical with *Endothia parasitica.*

Spaulding, P.

1912 Notes upon tree diseases in the eastern States. Mycologia 4:148–149.
Gives the distribution of the disease in Maryland, and advocates the cutting-out method.

Stewart, F. C.

1912 Can the chestnut bark disease be controlled? Pennsylvania Chestnut Blight Conference. Stenographic rept., p. 40–45. Harrisburg, 1912.
Author discusses his views of the impracticability of the cutting-out method.

Stoddard, E. M., and Moss, A. E.

1913 The chestnut bark disease. Connecticut Agr. Exp. Sta. Bul. 178:1–19.
A bulletin of general information.

Stone, G. E.

1911 The chestnut disease (Diaporthe parasitica). Massachusetts Agr. Exp. Sta. Report of the Botanist, Ann. rept. 23:24–25.
Gives distribution in Massachusetts; believes the disease is favored by weakness of the trees produced by unfavorable weather conditions.

Studhalter, R. A.

1914 Insects as carriers of the chestnut blight fungus. Phytopath. 4:52.
Abstract of paper read before the American Phytopathological Society, December, 1913. Author finds spores on twenty-four out of seventy-five insects taken from diseased trees.

Walton, R. C.

1914 The relation of temperature to the expulsion of ascospores of Endothia parasitica. Phytopath. 4:52.
Abstract of paper read before American Phytopathological Society, December, 1913. No ascospore ejection during the winter of 1912–1913; laboratory tests show that ejection is dependent on temperature.

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